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SYMPATHETIC INNERVATION OF THE NOSE

RESEARCH REPORT

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The results of anatomic and physiologic investigation indicate that the nasal membranes have a rich innervation from several sources. These nerves may be divided for our present purpose into two main groups—afferent and autonomic. The olfactory nerves are omitted from consideration.

The autonomic fibers come in part from the superior cervical ganglion through the internal carotid plexus. These are the thoracolumbar autonomic, or sympathetic, fibers. Others come from the great superficial petrosal nerve, synapsing in the sphenopalatine ganglion. They include vasodilator and secretomotor fibers. These are the cranial autonomic, or parasympathetic, fibers. The sympathetic fibers from the superior cervical ganglion are chiefly vasoconstrictors, but they also include vasodilators, according to Dastre and Morat.[1] The preganglionic fibers arise in the lower cervical and the upper thoracic portion of the cord, reaching the ganglion through the cervical sympathetic trunk.

Vasodilator and secretory fibers from the sphenopalatine ganglion to the nasal mucosa have been described by various investigators. Tschellussow [2] described a vasomotor path to the nasal membranes, with preganglionic fibers from the nerve of Wrisberg to Meckel's ganglion and postganglionic fibers from this ganglion to the nasal membranes. Blier [3] has shown that the vidian nerve and the posterior nasal nerves convey both vasoconstrictor and vasodilator fibers to the nasal mem-

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Research authorized and financed by the Council of the American Academy of Ophthalmology and Otolaryngology, from the Departments of Anatomy and Otolaryngology, University of Oregon Medical School.

1. Dastre, A., and Morat, J. P.: Recherches expérimentales sur le système nerveux vaso-moteur, Paris, G. Masson, 1884.

2. Tschellussow, M. A.: Die Innervation der Gefässe der Nasenschleimhaut, Arch. f. d. ges. Physiol. **151**:523, 1913.

3. Blier, X.: Physiology of the Sphenopalatine Ganglion, Am. J. Physiol. **93**:398, 1930.

branes. He did not differentiate the two parts of the vidian nerve—the great superficial petrosal and the great deep petrosal nerve. Electrical stimulation of the vidian nerve would affect both, with confusing results. Blier, however, obtained only vasoconstriction on stimulating the vidian nerve after applying nicotine to the sphenopalatine ganglion, when he waited long enough for the drug to produce its narcotic effect. The immediate effect of the nicotine itself was a transitory vasodilatation, indicating stimulation of the cells of the sphenopalatine ganglion, the fibers of which constitute the vasodilators. The vasoconstrictor effect of stimulation of the vidian nerve was lost when the superior cervical ganglion was removed and sufficient time was allowed for degeneration of the fibers from these cells. Obviously the vasoconstriction was due to stimulation of fibers from the superior cervical ganglion to the deep petrosal part of the vidian nerve.

It should therefore be emphasized that the vidian nerve consists of two functionally distinct parts, derived from widely separated regions of the nervous system (figs. 1 and 3). This has been pointed out on anatomic grounds.[4] The evidence from physiologic experiments should serve to make the facts even clearer. It is necessary to specify which of these parts, the great superficial petrosal nerve, or the great deep petrosal nerve, is concerned if one would correctly interpret phenomena due to involvement of the vidian nerve.

Jung, Tagand and Chavanne [5] and others have shown that the glands of the nasal region and the palate are innervated by postganglionic fibers from Meckel's ganglion. There is lack of unanimity, as well as of clarity, in the writings of different authors with regard to the course of the preganglionic secretomotor fibers. The preponderance of evidence indicates the great superficial petrosal nerve as the path.

No physiologic differentiation has been made between the vasodilators from the superior cervical ganglion and those from the sphenopalatine ganglion. Evidently the vasoconstrictor element of the superior cervical ganglion is much more important than the vasodilator element. Yet Dastre and Morat [1] and also Jung, Tagand and Chavanne [5c] have adduced seemingly reliable evidence that vasodilators ascend from the superior cervical ganglion. There is, however, no evidence of vasoconstrictor fibers in the great superficial petrosal nerve which relay in the sphenopalatine ganglion.

With this background of anatomic and experimental work, it is suggested that the clinical picture of congestion of the nasal membranes

4. Larsell, O., and Fenton, R. A.: Embryology and Neurohistology of Sphenopalatine Ganglion Connections, Ann. Otol., Rhin. & Laryng. **42**:739, 1928.

5. Jung, L.; Tagand, R., and Chavanne, F.: (*a*) Sur l'innervation excito-sécrétoire de la muqueuse nasale, Compt. rend. Soc. de biol. **95**:835, 1926; (*b*) Sympathique cervical et sécrétion nasale, ibid. **97**:94, 1927; (*c*) Sur la rôle trophique des nerfs sécrétoires, ibid. **99**:515, 1928.

associated with intestinal stasis, pelvic congestion, etc., may have its physiologic explanation in an overflow of vasodilator impulses to the nasal membranes. Whether such an overflow would be through the sympathetic or the parasympathetic route is not clear. The anatomic pathway for the sympathetic route is obvious. It is more difficult to account anatomically for an overflow of stimuli causing pelvic parasympathetic congestion, for example, to the parasympathetic innervation

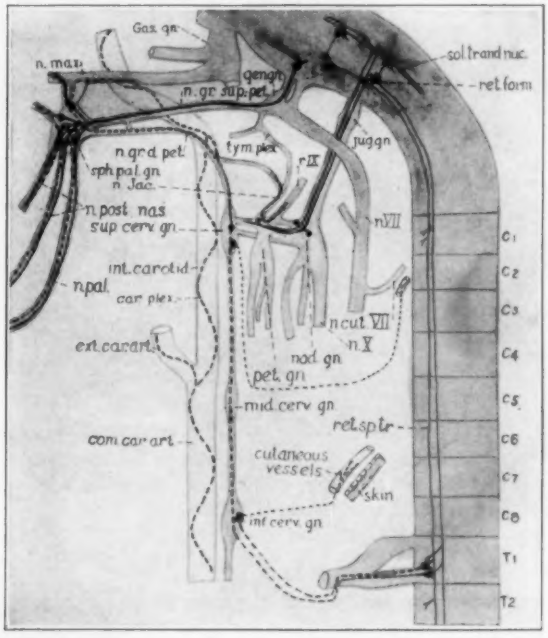

Fig. 1.—Sympathetic (broken lines) and parasympathetic (continuous lines) connections of the sphenopalatine ganglion and neighboring nerves. Part of the reticulospinal tract, the tractus solitarius and visceral afferent nerves into the latter are also shown. Visceral afferent fibers from the seventh nerve also enter the tractus solitarius, but are omitted for simplicity from the diagram (Larsell).

Abbreviations for all figures: *ch. tym.*, chorda tympani; *com. car. art.*, common carotid artery; *ext. car. art.*, external carotid artery; *Gas. gn.*, Gasserian ganglion; *gen. gn.*, geniculate ganglion; *inf. cerv. gn.*, inferior cervical ganglion; *int. car. art.*, internal carotid artery; *jug. gn.*, jugular ganglion; *lat. sp. thal. tr.*, lateral spinothalamic tract; *mid. cerv. gn.*, middle cervical ganglion; *n. cut. VII*, cutaneous branch of the facial nerve; *n. max.*, maxillary nerve; *n. gr. sup. pet.*, great superficial petrosal nerve; *n. gr. d. pet.*, great deep petrosal nerve; *n. Jac.*, nerve of Jacobson; *nod. gn.*, nodose ganglion; *n. pal.*, palatine nerve; *n. post. nas.*, posterior nasal nerves; *n. VII*, facialis nerve; *n. IX*, glossopharyngeal nerve; *n. X*, vagus nerve; *pet. gn.*, petrosal ganglion; *ret. form.*, formatio reticularis; *ret. sp. tr.*, reticulospinal tract; *r. IX*, root of glossopharyngeal nerve; *sol. tr.* and *nuc.*, tractus and nucleus solitarius; *sph. pal. gn.*, sphenopalatine ganglion; *sp. V tr.*, tract of fifth spinal nerve; *sup. cerv. gn.*, superior cervical ganglion; *tym. plex.*, tympanic plexus.

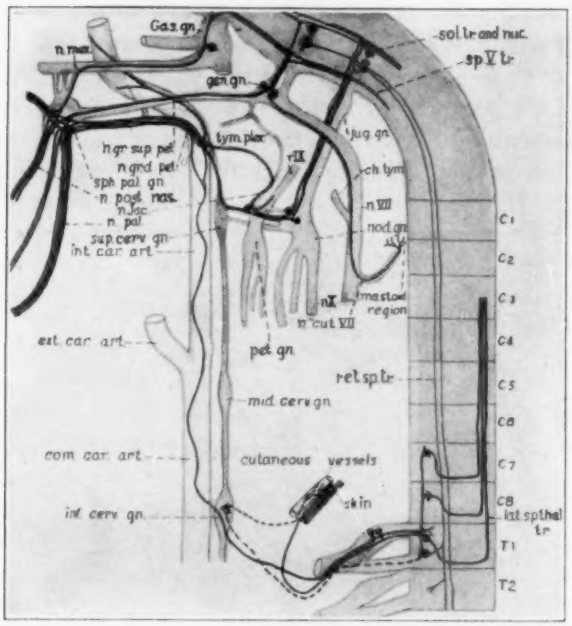

Fig. 2.—Afferent fibers in relation to the sphenopalatine ganglion. The cutaneous vasomotor reflex arc from the upper thoracic segments of the spinal cord is also shown, with the probable relation of segmental afferent fibers. The probable central connections are indicated (Larsell).

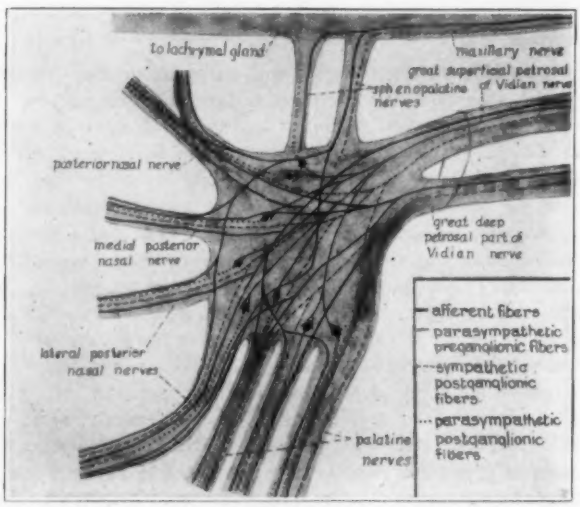

Fig. 3.—Sphenopalatine ganglion and its connecting fibers (Larsell).

of the nasopharynx. The pathway would necessarily lie in the cord, and there is no anatomic evidence for such a path. On the other hand, the abundant nasal discharge frequently accompanying nasal congestion associated with colonic or pelvic congestion suggests the cranial parasympathetic route, since secretomotor fibers belong to it. Such pathways and their relation to visceral organs must be taken into consideration if one would understand some of the phenomena of nasal physiology. The probable connection between exacerbation of residual sinus infection and such periods of vascular congestion in the nasal membranes is apparent.

The afferent fibers of the nasopharynx end chiefly in the mucous membrane itself, either in the epithelium or in the connective tissue beneath. It is probable that some here, as elsewhere in the body, end in relation to the blood vessels of the nasal mucous membrane. Like other nerve fibers, these undoubtedly respond to a sufficient stimulus anywhere along their course.

The afferent fibers are derived in large part from the gasserian ganglion of the trigeminal nerve. There is evidence, however, that afferent fibers arising in the geniculate ganglion, in the nodose ganglion and possibly some from the petrosal ganglion reach the nasopharynx and the palate (fig. 2). Kuntz [6] has adduced experimental evidence indicating that afferent fibers from the upper thoracic nerves reach the common carotid artery. Through the common carotid plexus and the internal carotid plexus, these fibers reach the nasal membranes and other parts of the head. The evidence that the fibers are connected with the cells of the dorsal root ganglions is not conclusive, as Hinsey [7] has pointed out. Kuntz [6] sectioned the common nerve trunks distal to the junction of dorsal and ventral roots, thus creating the possibility of ventral root fibers' being involved in the resulting degeneration. Hinsey's suggestion that the myelinated fibers which degenerate as a result of this operation may be preganglionic fibers to scattered ganglion cells along the course of the carotid artery and its branches must be admitted as a possibility.

The evidence, clinical, histologic and experimental, points for the most part toward the interpretation made by Kuntz. The clinical studies of Temple Fay [8] receive their most rational interpretation on this basis.

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6. Kuntz, A.: Nerve Fibers of Spinal and Vagus Origin Associated with the Cephalic Sympathetic Nerves, Ann. Otol., Rhin. & Laryng. **43**:50, 1934.

7. Hinsey, J. C.: The Anatomical Relations of the Sympathetic System to Visceral Sensation, A. Research Nerv. & Ment. Dis., Proc. **15**:105, 1934.

8. Fay, Temple: Atypical Facial Neuralgia: A Syndrome of Vascular Pain, Ann. Otol., Rhin. & Laryng. **41**:1030, 1932.

To this evidence we can add the results of the study of serial sections, stained by the Pal-Weigert method, of the human internal carotid artery and its nerve plexus, including the connections of the latter with the great deep petrosal and other neighboring nerves.[9] These studies, while not so detailed as those of Christensen [9a] on the cat owing to lack of sufficient material and limitations of technic on human material obtained at autopsy, agree, so far as they go, with the results obtained by Christensen.

Myelinated fibers of large and medium size are found in the internal carotid plexus in man. Reconstructions from the series stained by the Pal-Weigert method also show large myelinated fibers in one of the connecting filaments between the nodose ganglion and the petrosal ganglion. We have also found such fibers in the rabbit in serial sections stained with osmic acid. Myelinated fibers of similar size and appearance continue into the nerve of Jacobson. Part of this bundle branches into the lesser superficial petrosal nerve, part into the superior caroticotympanic nerve and part into the internal caroticotympanic nerve. Whether or not they all have their origin from cells of the petrosal ganglion, it is impossible to determine. Some may come from the nodose ganglion through the connecting bundle already described. It is not possible, however, to trace a continuity between the latter and the fibers of Jacobson's nerve among the cells of the petrosal ganglion.

Small myelinated fibers, corresponding in size to preganglionic fibers elsewhere, were present in our serial sections from the area between the petrosal ganglion and the superior cervical ganglion stained by the Pal-Weigert method. They passed from the latter into the carotid plexus. It did not appear probable that they were preganglionic fibers in this case. We also observed large myelinated fibers from the superior cervical ganglion to the carotid plexus and the great deep petrosal nerve. They were lost in the superior cervical ganglion, but it was evident that they did not extend into the cervical sympathetic trunk. It is probable that they bridged across to the petrosal or nodose ganglion, but definite histologic evidence was lacking in our material.

If the small myelinated fibers to the petrosal ganglion and the large fibers probably passing to the petrosal and nodose ganglions have cells in these ganglions, there is anatomic basis for the degeneration of fibers in the carotid plexus when the superior ganglion is removed, as observed by Kuntz in the cat. He did not state the size of the fibers

9. Larsell, O., and Burns, E. M.: Some Aspects of Certain of the Cranial Nerves, Ann. Otol., Rhin. & Laryng. **40**:661, 1931.

9a. Christensen, K.: The Innervation of the Nasal Mucosa with Special Reference to Its Afferent Supply, Ann. Otol., Rhin. & Laryng. **43**:1066, 1934.

which degenerated. It would also explain the results obtained by Davis and Pollock [10] of pain in animals from stimulation of the superior cervical ganglion.

Myelinated fibers were present in our human material in the carotid plexus below the level at which the great deep petrosal nerve joins it. Whether these continue to the upper thoracic dorsal root ganglions in man we have no means of ascertaining at present. The clinical observations of Temple Fay give some basis for assuming that they do.

In connection with the problem of referred pain from the nasal region another group of afferent fibers, not directly connected with the nasal mucosa, must be considered. These are the cutaneous branch of the facial nerve, the great auricular nerve and the cutaneous nerves to the neck and shoulder. Possibly deeper afferent nerves are also involved in so-called referred pain. The nerves named supply the skin of the mastoid region and of part of the head and neck. Various explanations of referred pain have been offered by Head, MacKenzie and others. If the general idea of an overflow of stimulation from visceral sources within the central nervous system eventually affecting cutaneous pathways which somehow project the sensation to corresponding segmental surfaces of the body is accepted, then the relation between afferent fibers in the great superficial petrosal nerve to fibers entering the medulla oblongata from the cutaneous branch of the seventh nerve is sufficiently close to explain referred pain in the mastoid region. Likewise the afferent fibers from the nasal region to the petrosal or nodose ganglion, the central roots of which must enter the bulb with cutaneous fibers of the ninth and tenth nerves, supply the connections necessary to account for basal headaches. The pain in the shoulder and arm, of which some patients complain, however, must be accounted for on a segmental basis in some other way.

The recent tendency is to explain so-called referred pain as due to vasomotor reflexes in the segment affected, producing changes in the skin, either chemical or ischemic. These cutaneous changes are then considered as giving rise to painful impulses directly involving the cutaneous afferent nerves of the segment affected. The fact that local anesthesia applied to such regions relieves pain lends great weight to this explanation and makes it appear as the simplest and most plausible yet offered.

In a series of seven patients with involvement of the sphenopalatine region and complaining of pain over the mastoid region, with symptoms typical of conditions which respond favorably to cocainization of the

10. Davis, L., and Pollock, L. J.: The Rôle of the Sympathetic System in the Production of Pain in the Head, Arch. Neurol. & Psychiat. **27**:282 (Feb.) 1932.

sphenopalatine ganglion region, we injected a 1 per cent solution of procaine hydrochloride into the skin between the ear and the mastoid process. This procedure was immediately followed by cessation of pain.

These results confirm those of Weiss and D. Davis [11] as well as those of L. Davis and Pollock.[12] They point to reflex vasomotor disturbances giving rise to pain in the mastoid region by involving the cutaneous branch of the seventh nerve and possibly the great auricular nerve. It is difficult, however, to account for so restricted a localization of vasomotor effects. Perhaps the thinness of the soft tissues above the mastoid process, with its correspondingly small blood supply normally, makes this region the first to be sufficiently affected to produce painful sensations when reflex ischemia is brought about by irritation of the sphenopalatine region.

The pathway from the source of irritation in the sphenopalatine region, described by Kuntz as reaching the upper thoracic segments of the cord by the great deep petrosal nerve and the carotid plexus, would be a favorable one for producing reflexes in this part of the cord. Vasomotor impulses originating here would reach the superior cervical ganglion, where they would be relayed to the postganglionic fibers of the carotid plexus which reach the posterior auricular artery and its branches. In basal headache and in pain involving the neck, shoulder and arm, associated with disturbance in the sphenopalatine region, a larger spread of the impulse within the cord so as to include vasoconstrictor nerves to these regions would be necessary. The afferent segmental nerves would be affected and through their connections with the spinothalamic tract would produce conscious pain.

There is another path by which impulses from the sphenopalatine region may reach the segments of the cord necessary for vasoconstrictor effects in the region under discussion. This is the reticulospinal tract, having its origin in the brain stem and ending at various levels of the spinal cord (figs. 1 and 2). Allen [13] has shown that this tract carries

11. Weiss, S., and Davis, D.: The Significance of the Afferent Impulses from the Skin in the Mechanism of Visceral Pain: Skin Infiltration as a Useful Therapeutic Measure, Am. J. M. Sc. **176**:517, 1928.

12. Davis, L., and Pollock, L. J.: The Rôle of the Autonomic Nervous System in the Production of Pain, J. A. M. A. **106**:350 (Feb. 1) 1936.

13. Allen, W. F.: (*a*) Formatio Reticularis and Reticulospinal Tracts: Their Visceral Functions and Possible Relationships to Tonicity and Clonic Contractions, J. Washington Acad. Sc. **22**:492, 1932; (*b*) Effect on Respiration, Blood Pressure, and Carotid Pulse of Various Inhaled and Insufflated Vapors When Stimulating One Cranial Nerve and Various Combinations of Cranial Nerves: I. Branches of the Trigeminus Affected by These Stimulants, Am. J. Physiol. **87**:319 (Dec.) 1928.

vascular reflexes. He has also demonstrated that stimulation of the trigeminal nerve in the nose or of the central stump of the severed vagus produces vasoconstriction and rise of blood pressure. Stimulation of the afferent fibers of the facial nerve from the nasal region or of fibers of the glossopharyngeal nerve, if these are present as suggested, would undoubtedly have the same effect. These would be visceral afferent fibers and so would end centrally in the solitary tract and its nucleus. Allen and Clark [15] have pointed out the pathway of reflexes from the solitary tract to the spinal cord, namely, by way of the cells of the nucleus of the solitary tract to the reticulospinal tract and through it to the cord. Here connections are made with the preganglionic fibers of the sympathetic vasoconstrictors emanating from the lower cervical and upper thoracic portion of the cord. The superficial vessels of the mastoid region and the superficial and possibly some of the muscular vessels of the base of the head, neck, shoulder and arm could thus be affected. The area involved would probably be in rough proportion to the degree of involvement of the sphenopalatine region, although it would no doubt vary with the individual subject also.

This suggested pathway, as well as that suggested by Kuntz, should be susceptible of experimental study. In either case the possible vasoconstrictor reflex arcs, together with the seventh, eighth and ninth cranial and the cervical nerves, offer an explanation of so-called referred pain from the sphenopalatine region which has much experimental and anatomic evidence in its favor. The hypothesis that vasoconstriction is reflexly produced by irritation of visceral afferent nerves offers a basis for understanding the beneficial effects of surface massage in instances of basal headache and related conditions.

The Council of the American Academy of Ophthalmology and Otolaryngology gave their continued support for our research.

14. Allen, W. F.: Experimental Anatomical Studies on the Visceral Bulbo-Spinal Pathway in the Cat and Guinea Pig, J. Comp. Neurol. **42**:393, 1927.

15. Clark, S. L.: Innervation of the Choroid Plexus and the Blood Vessels Within the Central Nervous System, J. Comp. Neurol. **60**:21, 1934.

LYMPHATIC PATHWAYS FROM THE NOSE

RESEARCH REPORT

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In a series of papers beginning in 1919 Mullin [1] has repeatedly called attention to the relation between drainage from the paranasal sinuses and certain types of pulmonary infection. He pointed out that particles of india ink or bacteria introduced into the sinuses of cats and rabbits, the sinus membranes being injured to facilitate absorption, are taken up by the lymphatics and find their way to the bronchial and mediastinal lymph nodes. Mullin and Ryder [2] reported that the inhalation of suspensions of carbon or of tubercle bacilli by rabbits results in pigmentations or tuberculous lesions similar to those of human tuberculosis, including involvement of bronchial lymph nodes.

Various clinical observers have emphasized one or the other of the possible routes of entry of infectious material to the lungs and the related lymph nodes in relation to bronchitis, bronchiectasis and other pulmonary disturbances. Quinn and Meyer,[3] Clerf [4] and others have pointed out the significance of aspirated material from the sinuses.

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1. Mullin, W. V.: The Lymph Drainage of the Accessory Nasal Sinuses, Tr. Am. Laryng., Rhin. & Otol. Soc. **25**:73, 1919; The Relation of Sinus Disease to Disease of the Chest, Tr. Mid-West. Sect. Am. Laryng., Rhin. & Otol. Soc. **26**:488, 1920; The Relation of Sinus Infection to Disease of the Lower Respiratory Tract, J. A. M. A. **87**:739 (Sept. 4) 1926; A Review of Sinus Chest Infections, Ann. Otol., Rhin. & Laryng. **41**:794, 1932.

2. Mullin, W. V., and Ryder, C.: Experimental Lesions of the Lungs Produced by the Inhalation of Fluids from the Nose and Throat, Am. Rev. Tuberc. **4**:683 (Nov.) 1920.

3. Quinn, L. H., and Meyer, O.: The Relationship of Sinusitis and Bronchiectasis, Arch. Otolaryng. **10**:152 (Aug.) 1929.

4. Clerf, L. H.: Bronchiectasis Associated with Diseases of Nasal Accessory Sinus: Etiology and Bronchoscopic Treatment of Bronchiectasis, Arch. Otolaryng. **6**:28 (July) 1927.

Lawson,[5] Mullin and others have written on the significance of the blood stream and the combined lymphatic system and blood stream as routes of entry.

Too little attention has been paid in all these studies to the defense mechanism of the lung itself and to the arrangement of the lymphatics and the lymphoid tissue therein in relation to the possible pathways of entry of particulate or infectious material. The beautiful and detailed studies of W. S. Miller[6] on the lymphatics of the lung give a firm basis on which to build a more complete knowledge of the pathways of infection in the lung and the neighboring structures.

In recent years much attention has been given to the lining of the pulmonary alveoli. Histologists are divided between the conception of an entodermal epithelial lining of the air sacs and the views advanced by Fried[7] and others that the lining is made up of mesodermal phagocytic cells closely related to if not identical with the histiocytes and macrophages elsewhere in the body. According to some views, this lining is not continuous. Rose[8] stated that the pulmonary capillaries themselves form the immediate lining of the air spaces, the latter thus corresponding to an open wound. Loosli[9] has presented evidence that the capillaries are uncovered. Miller[10] and others, on the other hand, have provided new evidences of a layer of flattened cells between the capillaries and the alveolar spaces. Fried has strongly emphasized the protective function of the alveolar surface against bacterial invasion.

Without attempting to determine the definitive structure of the alveolar wall, we shall present experimental evidence in the following description regarding the portals of entry of particulate matter into the lungs and the pathways of drainage from the paranasal sinuses and related regions into the chest. It will be attempted to correlate the evidence presented with the recognized facts of pulmonary anatomy.

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5. Lawson, L. J.: The Role of Nasal Accessory Sinus Membranes in Systemic Infection and Toxemias, Ann. Otol., Rhin. & Laryng. **39**:159, 1930.

6. Miller, W. S.: The Lymphatics and Lymph Flow of the Human Lung, Am. Rev. Tuberc. **3**:193, 1919; A Study of Tubercular Lesions in the Lung of a Negro Child Nine Weeks Old, ibid. **18**:373, 1928.

7. Fried, B. M.: The Lungs and the Macrophage System, Arch. Path. **17**:76 (Jan.) 1934.

8. Rose, S. B.: The Finer Structure of the Lung, with Special Reference to Its Vascular Character and Its Pathologic Significance, Arch. Path. **6**:36 (July) 1928.

9. Loosli, C. G.: The Rabbit's Lung After Phrenicotomy and Pneumothorax, Anat. Rec. (supp.) **61**:32 (March) 1935.

10. Miller, W. S.: The Epithelium of the Lower Respiratory Tract, in Cowdry, E. V.: Special Cytology, ed. 2, New York, Paul B. Hoeber, Inc., 1932, vol. 1, p. 105.

MATERIAL AND METHODS

Eighteen adult cats and three kittens were used in the experiments to be described. They were anesthetized with pentobarbital sodium given intraperitoneally, or with dial given intramuscularly. These anesthetics were used in preference to ether in order to prevent the large amount of mucous secretion in the air passages which results from the use of ether in cats and also because of the greater ease of administration. The adult animals were kept under the anesthetic for periods varying from thirty minutes to fifty-five hours before they were killed. During these various periods the lymphatics related to the upper respiratory tract were subjected to a continuous flow of the masses used or to repeated injections of them at intervals of from two to twelve hours.

The masses employed for injection were of three different types—prussian blue, trypan blue and potassium ferrocyanide. Prussian blue is usually employed for injection into the lymphatics. Ordinarily it is used as a suspension in a thin solution of warm gelatin. We used, however, a suspension of artist's prussian blue, obtained in tin tubes mixed with oil. This was dissolved in turpentine in the ratio of about equal parts of the mixture as it comes from the tube and turpentine. This suspension was then diluted with from four to five volumes of ether and injected with a no. 25 hypodermic needle and a Luer syringe. The needle was pushed through the soft palate and into the posterior wall of the pharynx at various points. Some of the mass escaped into the pharynx, whence it was removed with a swab. Most of it reached the lymphatic spaces and vessels, lymph nodes and fascial spaces of the retropharyngeal and cervical regions, as described later. Supplementary injections into the cervical lymph nodes, to fill their efferent lymphatics, were made by puncture of the nodes with the needle. After from ten to fifteen minutes of injection with this mass the animals died, probably from the effect of the ether superadded to the pentobarbital sodium already administered. They were then dissected and placed in a 10 per cent dilution of solution of formaldehyde U. S. P. The prussian blue was used to ascertain the gross distribution of the lymphatic vessels.

A method which was designed to demonstrate the finer lymphatics and lymph spaces and which, it was hoped, would make unnecessary the puncture of the lymph nodes to demonstrate their efferent lymphatics was used as follows: A 3 per cent solution of potassium ferrocyanide was injected into the frontal sinus or into the posterior wall of the pharynx, under slight pressure, for periods up to fifty-five hours. This substance is relatively nontoxic and produced no ill effect, so far as could be determined, on the animals anesthetized with pentobarbital sodium or with dial. After various periods the animals were killed; the bodies were dissected as much as necessary, and the head, neck and thorax, with the organs exposed, were placed in 5 per cent ferric citrate in a 10 per cent dilution of solution of formaldehyde U. S. P. The ferric citrate precipitated prussian blue from the potassium ferrocyanide in the lymphatics, lymph nodes and other structures which the injected fluid had reached. The solution of formaldehyde served to fix and preserve the tissues. To prevent the entrance of potassium ferrocyanide into the air passages and lungs during injection a tracheal cannula was inserted at the beginning of most of these experiments. When the animal was killed, the lungs were filled with fixing fluid through the cannula.

A third procedure consisted in introducing a solution of trypan blue in a physiologic solution of sodium chloride under the mucosa of the frontal sinuses or posterior pharyngeal wall. It will be recalled that trypan blue forms a colloidal solution, the particles of which are ultramicroscopic. Only when stored in phagocytic cells do the particles accumulate sufficiently to become visible with the micro-

scope. Two methods were used with the frontal sinuses. One consisted in exposing the bony wall above the eye, chipping away the bone to expose the membrane and then fixing needles in position between the membrane and the bone with bone wax. The needles, attached to flexible rubber tubing, were then connected with a funnel containing trypan blue. The solution was allowed to drain into the membrane of the sinus at the pressure produced by raising the funnel 50 mm. above the animal. A second method consisted in inserting small glass cannulas through small corks fitted into trephine openings in the bony roof of the frontal sinus and allowing the solution to enter the sinus itself under pressure similar to that already mentioned during a period of many hours. The ostium of the sinus had previously been plugged with cotton wool and bone wax.

To inject the material into the posterior pharyngeal wall, needles of suitable length and size were thrust through the soft palate and into the membrane of the pharynx. Adult cats under anesthesia and fitted with tracheal cannulas were given injections at intervals of about two hours for from twenty-four to fifty-five hours. Two kittens, anesthetized for the first injection, were given injections two or three times per day for eight and one-half and fourteen days, respectively. These did not have tracheal cannulas; so some of the trypan blue reached the trachea and probably the lungs by overflow dripping from the pharynx. A third kitten was subjected to repeated injections of trypan blue directly into the trachea for four days.

Sections were made of cervical lymph nodes, the carotid sheath, the esophagus and portions of the neck, lung, bronchial and mediastinal lymph nodes, etc. They were stained with orange G or with safranin. The latter stain has the advantage of staining both the nuclei and the cytoplasm.

OBSERVATIONS

The lymph drainage from the nasopharynx in the cat is chiefly toward the retropharyngeal and laryngeal region. Three cats given repeated injections of potassium ferrocyanide under pressure into the nostrils over a period of two hours showed an abundant precipitate of prussian blue in the cortex and some in the medulla of the deep cervical lymph nodes. The small lymph nodes, follicles and spaces of the retropharyngeal region also showed precipitate, and a small amount was present in the region of the palatine tonsil, especially in the spaces and channels of the muscle behind the tonsil. These cats were each fitted with a tracheal cannula, and the trachea was tied off anterior to the cannula to prevent the fluid from diffusing out in the visceral space. In two of these cats the tracheal lymph ducts showed precipitate.

Cat 9, which had potassium ferrocyanide injected into the frontal sinus for eight hours by the procedure described, showed blue precipitate in the deep cervical lymph nodes, the retropharyngeal region and the bronchial and mediastinal lymph nodes on microscopic examination. A tracheal cannula had not been thought necessary in this animal. The mucosa of the trachea and the bronchi showed considerable precipitate after fixation, indicating escape of potassium ferrocyanide into the air passages during the period of injection. The precipitate in the

bronchial, and possibly in the mediastinal, lymph nodes may therefore have been due to absorption of the injected solution by the lymphatics of the lungs.

It is clear from these experiments that the lymphatic drainage from the nasopharynx in the cat is primarily into the retropharyngeal and deep cervical lymph nodes. The drainage from the frontal sinuses is also into the retropharyngeal and deep cervical lymph nodes. The retropharyngeal nodes in the cat are small, most of the lymphoid tissue of this region being in the form of follicles rather than nodes. The normal membranes of the frontal sinuses are so thin as to leave little room for lymphatic vessels. There are, however, lymph spaces which gradually converge toward the main blood vessels supplying the sinuses. Around these vessels there are small lymphatic plexuses which drain into the retropharyngeal region and, directly or indirectly, into the cervical or laryngeal lymph nodes.

The efferent drainage from the laryngeal lymph nodes is through the tracheal trunks on the respective sides of the trachea. They drain into the corresponding jugular vein at its junction with the subclavian vein. These ducts receive tributaries from the posterior wall of the visceral space of the neck, as is shown by puncture injections of this wall at various points. These features are readily demonstrated by injections with the solution of prussian blue in turpentine if supplementary injections of the cervical lymph nodes are made to fill the larger ducts.

Injections of prussian blue into the posterior wall of the pharynx, using some pressure, result in an apparently continuous mass of blue coloring from the laryngeal region, posteriorly, on each side of the trachea, to the base of the neck. This continuous mass is formed by confluence of the tissue spaces in the loose areolar tissue along the esophagus and the trachea. The injected mass reaches these spaces from the tissue spaces of the pharyngeal wall when introduced into the retropharyngeal region and thus extends caudally to the anterior boundary of the chest. A similar but smaller mass of injected material extends downward in the carotid sheath.

In the fibrous coat of the posterior wall of the esophagus there are tissue spaces which extend, by continuity one with another, to the mediastinum. Injections of trypan blue show numerous macrophages along the length of the dorsal part of this tube as far as the upper part of the mediastinum (fig. 1). The lateral and ventral esophageal walls do not show such cells. Puncture injections of prussian blue into the dorsal wall of the visceral cervical space show some lymphatics which drain into the tracheal ducts, as stated, but a gentle, steady pressure on the syringe gradually fills tissue spaces which extend down-

ward toward the base of the neck. By puncture injection into the dorsal wall of the space in the base of the neck, about 1 inch (2.5 cm.) above the anterior wall of the mediastinum, the mediastinal tissue spaces dorsal to the heart and lungs were filled on each side. The injected mass extended well toward the bronchi and the hilus of the lung, on

Fig. 1.—A section from the dorsal wall of the mediastinal part of the esophagus of cat 16 after the repeated injection of trypan blue for fifty-five hours into the mucous membrane of the posterior portion of the nasopharynx. Some of the macrophages laden with trypan blue are indicated by arrows.

the one hand, and well toward the caudal part of the mediastinum, on the other hand.

Cats 11 and 12, which were fitted with tracheal cannulas and given injections of trypan blue for twenty-eight and twenty-two hours, respectively, by inserting a needle into the posterior wall of the pharynx,

showed trypan blue in sections of the laryngeal, small deep inferior cervical, bronchial and dorsal mediastinal nodes. In the latter there were numerous granules in both the peripheral and the deep sinuses and in large phagocytic cells in the nodes. The reticular cells of the cervical, bronchial and mediastinal nodes also showed ingested trypan blue. The septal and dust cells of the lungs and the pulmonary lymphatics and lymph follicles also showed the dye.

Cats 13 and 14, both kittens about 6 weeks old at the time of the initial injection, received doses of about 1 cc. of a 0.5 per cent solution of trypan blue by injection several times per day into the posterior pharyngeal wall for fourteen days and eight and one-half days, respectively. They suffered no untoward effects save for from five to ten minutes after each injection, during which time they were removing the overflow of the injection from the nasopharynx. Some of the excess undoubtedly dripped into the trachea and the lungs.

These kittens when killed and dissected showed substantially the same picture as the adult cats. The deep cervical or laryngeal nodes were strongly colored with trypan blue, visible grossly. The prevertebral fascia showed blue coloration, grossly, about midway to the base of the neck. The amount of coloration diminished downward, with but a small amount in the lower part of the region of the neck.

Microscopically, the upper part of the esophagus showed great numbers of blue-laden macrophages in the loose connective tissue of the dorsal wall and extending into the striated muscle layer along the connective tissue septums. Between the bands of muscle in the thinner septums blue-laden cells were numerous throughout the muscular layer. Sections from the connective tissue of the dorsal esophageal wall also showed small lymph channels, containing lymphocytes and blue-laden phagocytes. Evidently there was much diffusion of the injected solution downward from the posterior pharyngeal wall along the tissue spaces behind the esophagus. Migration of histiocytes and macrophages probably does not account for the widespread distribution of the trypan blue along the dorsal wall of the esophagus and caudally toward the thorax, since the lateral walls were almost free from blue granules in corresponding cells.

In a mediastinal lymph node dorsal to the esophagus, which was dissected out, together with neighboring adipose tissue and fascia, there were numerous blue-laden macrophages. Between the fascial layer and the fat there was a lymphatic vessel of considerable size containing numerous lymphocytes and dye-laden macrophages. This was probably an afferent lymphatic, as the mediastinal node described was regarded as having received its trypan blue from the mediastinal and lower cer-

vical lymph spaces, rather than from the lungs, by reason of its position and apparent connections.

Cat 16, which was fitted with a tracheal cannula and kept under anesthesia for fifty-five hours, was given repeated injections during this period of trypan blue under pressure into the posterior pharyngeal wall. The needle was kept attached in position between injections and was continuously filled with the solution.

Grossly the cervical lymph nodes were colored a deep blue by the dye. From the posterior pharynx a zone of deeply stained tissue extended along the prevertebral fascia into the mediastinum and the thorax, diffusing into the loose connective tissue on each side of the esophagus. Behind the esophagus there were definite lymphatic channels as far as the inferior cervical region. Within the upper part of the thorax the blue coloration extended beneath the parietal pleura for a short distance on each side. The dorsal mediastinal lymph nodes also appeared blue to the naked eye. The upper mediastinal portion of the esophagus showed lymph channels with blue coloration in the adventitial coat just outside the layer of muscle.

Microscopically, both the lower cervical and the mediastinal portion of the esophagus showed macrophages laden with trypan blue in the adventitial coat and also in the septums extending into the layer of muscle. Lymph channels containing lymphocytes and phagocytes laden with trypan blue were abundant. These discharge from the mediastinal portion of the esophagus into the mediastinal lymph nodes. The lower thoracic portion of the esophagus and the aorta showed no indication of the trypan blue. The superior mediastinal nodes showed peripheral sinuses distended with fluid and containing many macrophages laden with trypan blue. Such cells were less numerous in the deep part of the nodes. The carotid sheath, both upper and lower, had spaces filled with phagocytes laden with trypan blue. There were also definite lymphatic channels distended with fluid and containing phagocytes and lymphocytes. The prevertebral fascia of the base of the neck microscopically showed lymphatic vessels and loose connective tissue containing numerous phagocytes filled with trypan blue.

THE LUNGS AND BRONCHIAL LYMPH NODES

In a number of the experimental cats the bronchial lymph nodes showed precipitate from the treatment with potassium ferrocyanide and ferric citrate or granules of trypan blue. It was necessary to determine the pathway of entrance. There was some intercommunication between the tracheobronchial and the mediastinal nodes, but the bronchial nodes near the hilus of the lung are usually regarded as receiving their afferent lymphatics from the lungs. The detailed anatomic studies of W. S.

Miller [6] have provided complete information concerning the lymphatic drainage of these organs.

Cats 13 and 14 were given injections of trypan blue over relatively long periods without tracheal cannulas. There was therefore some dripping probably, as already stated, from the pharynx into the lungs. Yet the trachea and the bronchi were clean when the animals were killed several hours after the last injection. The lungs of these kittens were blue macroscopically, and on examination of sections they were seen to be filled with cells of various kinds, laden with granules of trypan blue. The air sacs contained numerous large cells, some free, others in various stages of detachment from the alveolar walls. Many of them contained trypan blue. The peribronchial and perivascular lymphatics in all parts of these lungs were distended with fluid and contained numerous phagocytic cells with granules of trypan blue (fig. 2). The lymphatics of the pleura likewise were distended and contained phagocytes. It will be recalled that the lymphatic drainage from the outer part of the lung is into the pleura.

We shall not enter here into a discussion of the lining of the air sacs. Histologists are divided into two schools, some holding the view that the alveoli of the lungs are lined with flattened entodermal cells, while others claim that the lining, so far as it is present, is mesodermal and related to macrophage cells. A recent brief discussion of the salient points of the opposing views is contained in the new (1936) edition of Stöhr's "Text Book of Histology," [11] to which reference is made.

The point of importance in this presentation concerns the pathway of entrance into the lung of the granules of trypan blue contained in the septal and dust cells and in the phagocytes within the lymphatics. Since the only lymphatics connected both with the lungs and with adjacent structures are the efferent channels at the hilus, there remain but two possible pathways of entry of the phagocytosed material—the trachea and the blood stream.

In cats 13 and 14 both these channels were open. Both these animals showed a large amount of phagocytosed material within the lungs. This may be due to the length of the experiment with them, to the fact that both channels of entry were open or to a combination of these two factors. It was easy to rule out the tracheal pathway by repeating the experiment on animals provided with trachea cannulas. A number of cats were so treated. It was thought desirable to keep them anesthetized during the experiment; so it was not practicable to continue the experiment longer than for from twenty-eight to fifty-five hours. The results were essentially the same in all, save for the quantity of trypan

11. Stöhr, P.: A Text Book of Histology, edited by J. L. Bremer, ed. 5, Philadelphia, P. Blakiston's Son & Co., 1936.

blue observed within the lungs. Since the treatment of cat 16 was continued for the longest period, namely, fifty-five hours, the more detailed description will be based on the results obtained with it. In this animal the lungs showed peribronchial and perivascular lymphatics filled with phagocytes laden with trypan blue (fig. 3). The pleural

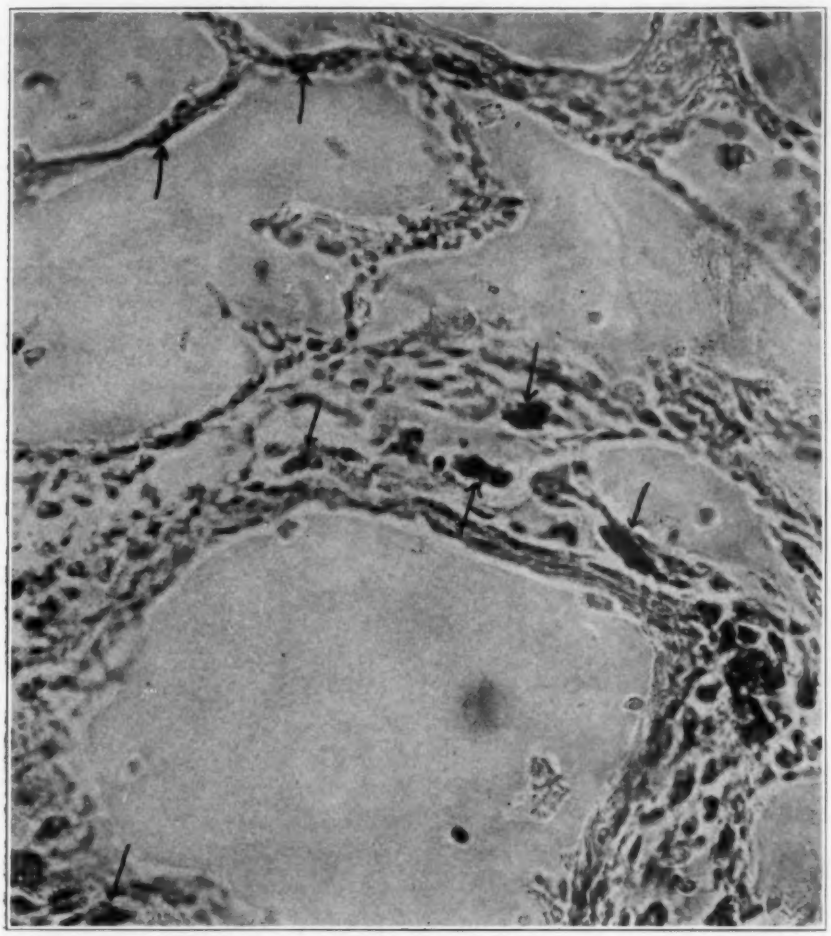

Fig. 2.—A section from the lung of kitten 14, showing cells laden with trypan blue in the perivascular lymphatics and septal cells containing particles of dye. This cat had trypan blue injected into the posterior wall of the pharynx at intervals for eight and one-half days. Some of the solution escaped into the trachea, probably at each injection.

lymphatics also contained such cells. Neither the pleural nor the intrapulmonary lymphatics were as distended with fluid as those in the kittens already described.

Septal cells and dust cells containing trypan blue were numerous, but the amount of blue in the individual cells and the size of the granules were less than in the kittens. This was the only difference clearly

Fig. 3.—A section from the lung of cat 16, which was provided with a tracheal cannula and given injections of trypan blue into the posterior wall of the pharynx repeatedly for fifty-five hours.

marked, although one gains the impression that the number of cells containing granules was not so great in the adult cats as in the kittens. This may be due to differences in the time involved, to the closure of

the tracheal portal of entry in the adult cats or to both factors. There is, however, no question of the presence of granules in the phagocytic cells of the cannulated cats.

The trypan blue could have entered the lungs only through the blood stream in these animals, taking its path from the retropharyngeal and cervical lymph nodes, through the tracheal lymph ducts to the external jugular veins, and thence to the right side of the heart and the pulmonary capillary bed. Diffusing through the endothelial cells, the colloidal particles were taken up by the septal cells. By repeated performance of this phagocytic function on the part of these cells, the ultramicroscopic colloidal particles gradually accumulated within the cells sufficiently to become visible granules. The size of the granules within the cells in animals treated for different periods was roughly in proportion to the period of phagocytic activity in ingesting the trypan blue. They were largest in the animals subjected to injections for the longest period. Particles of trypan blue were found along the capillary bed in general, but also in detached cells in the alveoli. The endothelial cells are in such close juxtaposition to the flattened cells, whether they are epithelial cells or macrophages, which cover the capillaries, that the technic employed is inadequate to determine whether such particles are contained within them or not. The preponderance of evidence from our preparations was negative. Foot,[12] who has made a special study of phagocytic activity by the endothelium of the pulmonary bed, recently revised his earlier position and stated that it does not have phagocytic properties, a view shared by Wislocki.[13]

It is generally accepted that the so-called "dust cells" phagocytose particulate material brought to the alveoli with the air in breathing. Fried held that the lining cells of the alveoli are macrophages and that the alveolar wall "is a veritable fortress when bacteria have penetrated into the respiratory portion of the lungs." He found that infection by the pathway of the pulmonary blood stream is much more easily produced than that by the tracheal route. Wislocki has shown that carbon particles from 1 to 3 microns in diameter injected into the blood stream of rabbits and cats are first deposited in the lungs as capillary plugs. They are then gradually removed through escape into the general circulation, through lymphatics draining into regional lymph nodes and through phagocytosis by "clasmatocytes." The origin of the latter Wislocki was unable to ascertain.

12. Foot, N. G.: On the Origin of Pulmonary Dust Allergy, Am. J. Path. **3**:413, 1927.

13. Wislocki, G. B.: On the Fate of Carbon Particles Injected into the Circulation, with Especial Reference to the Lungs, Am. J. Anat. **32**:423, 1924.

We have seen a few capillary plugs of trypan blue in the lungs of our cats, but they appeared relatively unimportant. It seems more likely that the fine colloidal particles of this substance diffused through the endothelial cells as they reached the lungs, from the beginning of the series of injections to which our animals were subjected. In this

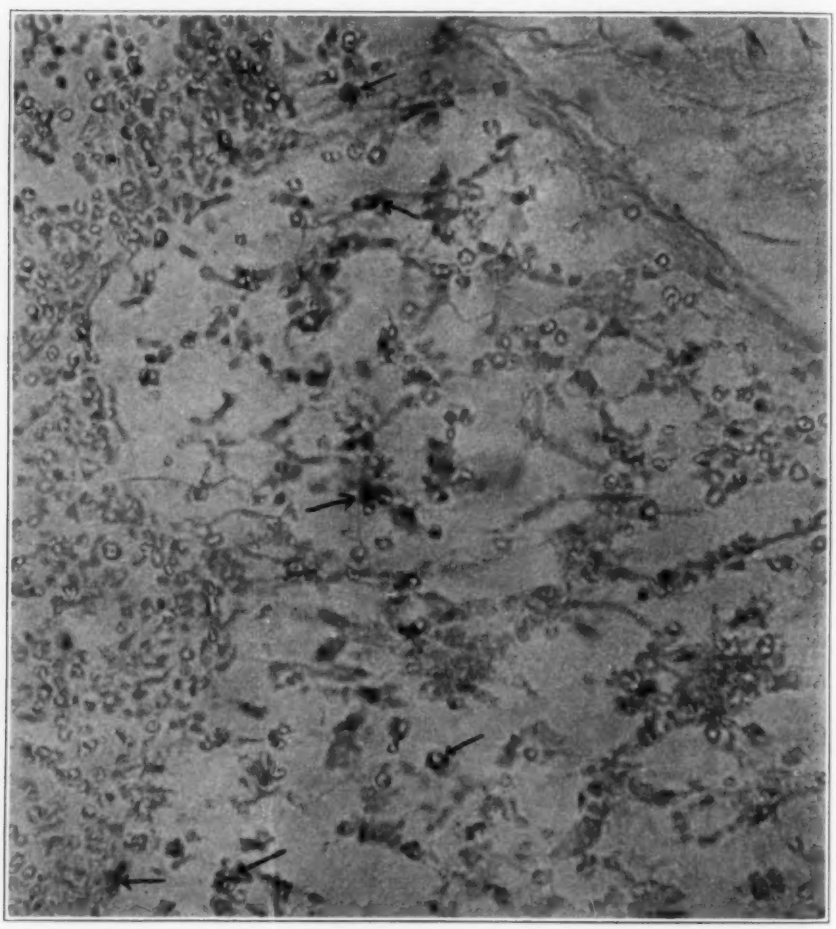

Fig. 4.—A section from an anterior mediastinal lymph node of cat 14, showing macrophages laden with trypan blue.

respect injections of trypan blue would differ from bacterial invasion of the lungs by the same route. Bacteria if present in sufficient number would probably form plugs as did the particles of carbon of approximately comparable size (streptococci, 0.6 to 1.5 microns). The method of removal by phagocytic cells is the same for inert particles of carbon

and trypan blue and undoubtedly for bacteria, save that the latter, through their proliferation and formation of toxic substances, present a more serious problem to the defense mechanism.

As already stated, the phagocytes laden with trypan blue leave the lung through the pulmonary lymphatics. Some of these drain directly to the hilus of the lung, accompanying the walls of the bronchi or the

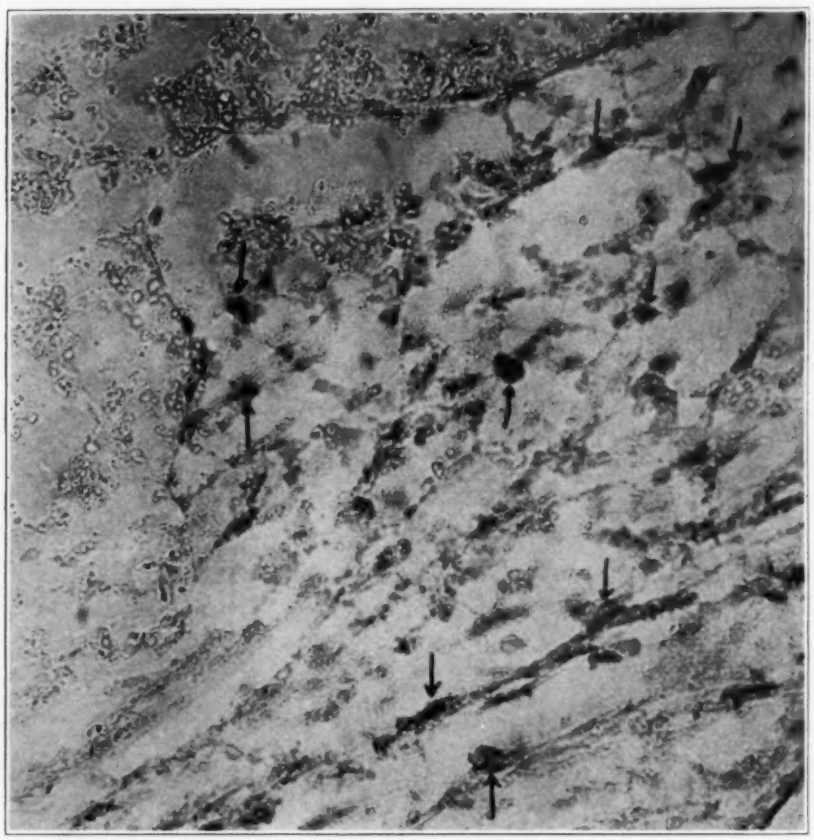

Fig. 5.—A section from an anterior mediastinal lymph node of cat 16, showing macrophages laden with trypan blue.

blood vessels. From the outer portion of the lungs the drainage is first to the pulmonary pleura and then to the hilus through the pleural lymphatics. By either pathway the pulmonary stream passes eventually into the tracheobronchial lymph nodes and neighboring nodes in the mediastinum. In all our animals in which the lungs showed trypan blue, these lymph nodes were colored with the dye (figs. 4, 5 and 6).

Some of the phagocytic cells also escaped through the air passages. "Dust cells" laden with trypan blue were encountered free in alveoli in all parts of the lung, even those just beneath the pulmonary pleura. In some of the cats which had been cannulated such cells were found

Fig. 6.—A section from a tracheobronchial lymph node of cat 16, showing macrophages laden with trypan blue.

in considerable numbers mixed with mucus in the bronchi. In many of the smaller bronchi of cat 11, in spite of the tracheal cannula, the mixed mass of cells and mucus almost closed the lumen. In the larger bronchi they lay against the wall, enclosed by mucus. The presence of

dye in their cytoplasm indicated that they must have become detached from the septal walls before the animals were killed. They obviously were desquamated cells which would normally have been discharged with the mucus secretions through the trachea. Cat 21 was given injections on five successive days of about 1 cc. of trypan blue solution, introduced with a needle directly into the trachea below the larynx. The dye-containing dust cells were about as numerous as in cats when the bronchial passages were guarded by a tracheal cannula. The septal cells with blue granules did not appear to be so widespread. Both the peribronchial and the perivascular lymphatics, however, were filled with phagocytes laden with trypan blue. The larger bronchi showed a considerable coating of trypan blue over the epithelial surface. The bronchial and tracheobronchial lymph nodes were blue macroscopically.

SUMMARY AND CONCLUSIONS

There are four routes by which material in solution or suspension can reach the bronchial and mediastinal lymph nodes from the region of the paranasal sinuses. These are (1) the trachea, (2) the combined path of the lymph nodes, tracheal lymph duct and blood vessels through the right side of the heart and the pulmonary bed, (3) the purely hematogenous path and (4) lymph spaces and channels in the visceral cervical space, the dorsal wall of the esophagus, the prevertebral fascia, and related structures, which communicate with the anterior part of the mediastinum. The pathways by which the trypan blue reached the chest from the paranasal sinuses and from the posterior portion of the nasopharynx are shown in the accompanying tabulation.

Pathway of Trypan Blue from Paranasal Sinuses to Chest by Lymphatic and Blood Stream Route

1. Sinus lymph spaces and small lymphatics
2. Retropharyngeal and deep superior cervical lymph nodes
3. Cervical lymphatics
4. Subclavian vein, superior vena cava, right side of the heart
5. Pulmonary capillary bed
6. Diffusion through capillary endothelium
7. Phagocytosis by septal cells, some of which become detached as alveolar "dust cells"
8. Migration of septal cells into the perivascular and peribronchial lymphatics in the interior of the lung and into the pleural lymphatics from the outer portion of the lung
9. Drainage of perivascular, bronchial and pleural lymphatics into bronchial and mediastinal lymph nodes; enlargement of lymph follicles along intrapulmonary bronchial walls
10. Alveolar "dust cells" discharged with mucus through the bronchial passages

Pathway of Trypan Blue from Posterior Nasopharynx by Tracheal Route

1. Trachea, bronchi, bronchioles
2. Air sacs
3. Phagocytosis by septal cells and alveolar "dust cells"
4. Septal cells laden with trypan blue migrate into the peribronchial and perivascular lymphatics and the bronchial lymph follicles; some migratory cells in pleural lymphatics
5. Drainage into bronchial and mediastinal lymph nodes

The tracheal route is obvious. It is particularly important in airborne infection. The experiments with trypan blue clearly indicate that drippings from the posterior pharyngeal wall and the nasopharynx also reach the lungs. The particulate matter, phagocytosed by septums and dust cells, is in part eliminated through the bronchial passages, but most of the phagocytic cells get into the perivascular, peribronchial and pleural lymphatics and thence into the bronchial and neighboring lymph nodes. Lymph follicles in the bronchial walls within the lung become enlarged and when bacterial invasion is involved no doubt become infected.

Colloid material reaching the lungs by the combined lymphatic and blood vascular route diffuses through the capillary walls to be phagocytosed by the same type of cells as serve for the tracheal route, namely, septal cells and the so-called dust cells. The pathway of these cells from the lungs is again through the various lymphatic channels of the lungs to bronchial and mediastinal lymph nodes. When particulate material of larger than colloidal size enters the pulmonary bed, there is formation of capillary plugs, with subsequent phagocytosis. The phagocytes so involved also can escape from the lungs only by the lymphatic channels already named. Bacterial invasion undoubtedly may form capillary plugs which in turn become centers of proliferation and of long-continued phagocytic activity, with long-continued irritation to the lymphoid tissue and the neighboring structures.

The purely hematogenous route of invasion from the region of the sinuses is possible, but appears unlikely. Aside from the mediation of lymph nodes and lymphatic vessels, the pathways would be the same as when the lymphatic route is involved. The pulmonary lymphatic pathways and cellular elements would, of course, also be the same.

The fourth route mentioned, namely, the lymph spaces and channels from the neck which communicate with the mediastinum, is probably of little significance. Bacteria escaping from the retropharyngeal region into adjacent tissue spaces are probably phagocytosed by the numerous histiocytes in the looser tissues before they have gone far. The communication of tissue spaces in the connective tissues continuous from

the neck to the thoracic wall and mediastinum, however, indicates the possibility of this pathway.

The lymphatic-blood vascular and the tracheal route were certainly the most important paths by which material from the sinuses entered the lungs and the related lymph nodes in the experiments reported. The evidence does not permit definite conclusions as to which of these two routes was the more important, but it appears to point toward the path of the combined lymphatics and blood stream.

After this paper was sent to press, my attention was directed to the evidence presented by Ravenel and Reichel [14] that tubercle bacilli may pass through the intestinal wall and reach the lungs by way of the thoracic duct and the right side of the heart. Although it has been repeatedly demonstrated that epithelium is resistant to trypan blue and related substances, the fact that our animals ingested through the esophagus considerable amounts of the dye during the course of an experiment made it appear desirable to examine this possible pathway of entry into the lungs.

A kitten was given 30 cc. of a 0.5 per cent solution of trypan blue by mouth daily for five days. The solution was thoroughly mixed with the ordinary animal house ration, so that virtually all of it, a total of 150 cc., was consumed. This kitten (*22*) was about the same age and size as kitten 21, which had been given intratracheal injections of a total of about 7 cc. of a solution of trypan blue also during a period of five days.

Kitten 22 passed blue feces from the first day of the experiment. It was killed on the fifth day, two hours after the last meal containing dye mixed with the food. Digestion was in full progress. When the abdomen was opened, there was no evidence of coloration by the dye, either of the wall of the digestive tube or of the lacteals issuing from it. The latter were plainly visible as white threads in the mesentery. The digestive tube itself, when opened, revealed a continuous mass of deep blue food and fecal matter throughout its length. Sections of the esophagus, stomach, duodenum, jejunum, ileum and colon, stained with safranin, gave no sign of absorption of trypan blue by these organs. Sections of lungs, liver, spleen and kidney likewise were free from granules of trypan blue. The intestinal route of absorption can therefore be ruled out in our experiments.

14. Ravenel, M. P., and Reichel, John: Tuberculous Infection Through the Alimentary Canal, J. M. Research **18**:1, 1908.

SUBSTANCES RESEMBLING EPHEDRINE IN EXPERIMENTAL AND IN CLINICAL SINUSITIS

RESEARCH REPORT

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After our report in 1935 [1] of the reparative reactions produced in acute experimental sinusitis in the cat by certain substances resembling ephedrine, Dr. K. K. Chen requested us to report on four of these drugs, especially as to their shrinking powers and their reparative effects in chronic inflammation of the sinuses.

SUMMARY OF EXPERIMENTS

In a considerable series of private patients the effects of these substances on the inferior turbinate were tested by swabbing on a 1 per cent solution, and in a smaller series the solution was dropped into the nose with the head reversed, with the following results:

Tetrandrine methiodide in a 1 per cent and in a 0.5 per cent solution produced a pinkish precipitate at room temperature after twenty-four hours; hence it was used only a few times. Shrinkage was rather marked, but some complaint was made of a bitter taste and slight burning.

Hordenine methiodide in a 1 per cent solution produced fair shrinkage, slight sneezing and burning and a rather persistent bitter acid taste.

Benzyldimethylamine methiodide in a 1 per cent solution gave excellent shrinkage, with little or no burning, but was slightly bitter.

Betaphenylethylamine in a 1 per cent solution produced fair shrinkage, with slight burning and a fairly bitter taste.

No evidence of systemic absorption or of change in pulse or blood pressure was noted.

Two middle-aged persons requiring radical maxillary operation for chronic infection received three injections of hordenine methiodide in

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Research requested and financed by Eli Lilly and Co., from the Department of Anatomy and Otolaryngology, University of Oregon Medical School.

1. Fenton, R. A., and Larsell, O.: Research Report on Experimental and Clinical Sinusitis, Arch. Otolaryng. **23**:18 (Jan.) 1936.

a 1 per cent solution and of betaphenylethylamine in a 1 per cent solution, and the tissues were removed at operation one day after the third injection. No clinical improvement was reported by the first patient, while the second reported great relief from congestion and was with difficulty persuaded to have an operation; no purulent exudate was found, and the chronically infected membranes were filled with plasma cells, with little or no evidence of reparative changes.

Cats in which the sinuses had been infected for six weeks received perfusions with the various solutions, which were also tested in a number of uninfected sinuses. Apparently all these solutions are somewhat irritating to normal ciliated sinus epithelium, especially betaphenyldiamine and tetrandrine methiodide.

Little or no evidence of active stimulation of repair could be found in most of these chronically infected membranes, although less damage to cilia was found with benzyldimethylamine methiodide, and more migrant leukocytes with betaphenylethylamine. Eosinophils were more noticeable with the use of tetrandrine methiodide and of betaphenylethylamine.

The value of these substances would seem to lie in their clinical use in relatively acute conditions, after determination of necessary pharmacologic factors, particularly for benzyldimethylamine methiodide and betaphenylethylamine.

DETAILS OF EXPERIMENTAL STUDIES

The experimental work was carried out with the technical assistance of Dr. Wallace Shearer in the department of anatomy. Surgical intervention on the maxillary antrum was done by Dr. W. N. Thompson, resident in the department of otolaryngology.

Three groups of cats were chronically infected with hemolytic streptococci, and the various 1 per cent solutions were perfused through their sinuses seventy-two hours before they were killed.

The chronic infections were produced as follows: The skin over the frontal sinuses was incised, with the animal under ether anesthesia. The subcutaneous tissues and the periosteum were scraped away, and then by the use of an antrum needle a small amount of semisolid 1 per cent agar was injected into the sinus, thus partially closing the ostium of the sinus. This was followed by the injection of about 1 cc. of a twenty-four hour culture of hemolytic streptococci obtained fresh from a patient in the hospital with a virulent infection. In about forty-eight hours in most cases the cat acquired a head cold with sneezing and a nasal discharge. This usually remained as an acute infection for about three weeks, sometimes longer. At the end of five weeks these animals were considered chronically infected, in accordance with previous microscopic studies.

In the first group of cats only the right sinus was infected, the left being normal. In this group both sinuses, the infected and the normal, were perfused with the solutions until the fluid flowed from the nostrils. In the second group also only the right sinus was infected, but in this case each animal received two

perfusions in each sinus twenty-four hours apart. In the third group both sinuses were infected, but only the right was perfused with the solutions.

In each case the animals were killed seventy-two hours after the final perfusion. Eight cubic centimeters of 1 per cent trypan blue was injected into the subcutaneous tissue of the neck at the time of the last perfusion.

Hordenine methiodide in a 1 per cent solution and betaphenylethyldiamine in a 1 per cent solution were used for the available human subjects. Two patients, each scheduled for radical removal of the membrane from the left maxillary sinus, were used. Six days previous to the operation the left antrum in each case was irrigated with water. At the end of the irrigation, after two injections of air to remove the water, 2 cc. of the solution was injected into the sinus and left there. This process was repeated twice more, injections being two days apart, and the operation was done the day after the final injection.

CAT 1-36.—*Right Sinus.*—There was chronic infection of the right frontal sinus. The reaction in this cat was not especially acute. A moderate discharge was noted during the first week. During the second week no discharge was present, and the cat was apparently well. For five days in the third week a profuse seropurulent discharge was noted, with apparent recovery in the following weeks. At the end of the sixth week the cat was anesthetized, and about 2 cc. of tetrandrine methiodide in a 1 per cent solution was perfused through the sinus (until it appeared at the nostril). Three days later the cat was killed; the head was cut off, and the sinus membrane was exposed. The specimen was placed in Zenker's fluid and solution of formaldehyde for twenty-four hours, washed for twenty-four hours and then placed in 70 per cent alcohol. At this stage, the sinus membrane was dissected from the skull. It was thickened and somewhat adherent and contained about 0.5 cc. of purulent material. No gross hyperemia was noted. The membrane was then carried through the higher alcohols and xylene and embedded in paraffin. It was then sectioned at 6 microns thickness and stained according to the Maximow hematoxylin-eosin-azure technic. Microscopically, most of this membrane was normal in appearance. The epithelium and the cilia were normal, save for some increase in goblet cells. The tunica propria was slightly thickened in parts. One area was considerably thickened, with an accumulation of lymphocytes and plasma cells resembling an old chronic infection of the membrane. Few leukocytes were present. Some of the blood vessels in section had many eosinophils. Some edema was present.

Left Sinus.—This sinus was not infected. One perfusion of tetrandrine methiodide in a 1 per cent solution was done at the same time as on the infected side, and the membranes were prepared in the same manner as those from the right sinus. In this case the membrane was not adherent. It was thin and glistening and was not hyperemic, except for evidence of a slight hemorrhage at the site of injection. No exudate was present within the sinus. Microscopically, the membrane showed evidence of a mildly acute irritation, with edema, fairly numerous polymorphonuclears, including eosinophils, and some lymphocytes. The epithelium was considerably injured in spots, some places being almost destroyed; elsewhere the cells appeared nearly normal, with intact cilia. There was a mucous cyst surrounded by low epithelium. The membrane was hyperemic. The entire picture pointed to considerable irritation by the injection of fluid.

CAT 2-36.—*Right Sinus.*—There was chronic infection of the right frontal sinus. This cat was sick for two days following infection. A nasal discharge was present for three weeks. The cat was in excellent spirits and of excellent appearance after this time. One cubic centimeter of hordenine methiodide in a 1 per

cent solution was perfused through this sinus at the end of the sixth week. Dissection showed a thickened dull membrane, not as adherent as that in the right sinus of cat 1 but containing a little more exudate and somewhat thinner. Some hyperemia of the membrane was noted. Microscopically, the membrane was greatly thickened, with considerable fibrosis in the deep part of the tunica propria. The outer region of the tunica propria was filled with lymphocytes and plasma cells and some polymorphonuclears. Considerable hyperemia was present. The surface epithelium consisted of elongated columnar cells much taller than normal, with numerous goblet cells. There were many mucous cysts in various stages of development extending into the tunica propria. The cilia appeared about normal in some parts of the epithelium; elsewhere they were clumped together, and in some places apparently destroyed. The blood vessels of the tunica propria contained numerous eosinophils. There were some large cystlike structures deep in the tunica surrounded by evidence of much destruction of tissue. Some parts of the membrane showed great concentration of the plasma cells beneath the epithelium but little evidence of acute exacerbation.

Normal Left Sinus.—About 1 cc. of hordenine methiodide in a 1 per cent solution was perfused through this sinus. An apparently normal membrane was noted on dissection. Slight hyperemia was present at the site of injection. Microscopically, the epithelium was normal in appearance; the cilia were intact; the cells were low columnar to cubical, with few goblet cells. The tunica propria was slightly edematous in appearance and slightly hyperemic. Injected material in the sinus cavity was filled with red blood corpuscles.

Cat 3.—This animal was prepared in the same manner as the two previously described. The reaction throughout was exceptionally mild. A definite infection was obviously present, however, as indicated by a persistent watery discharge from the cat's nose. The infected right side was successfully perfused with benzyldimethylamine methiodide in a 1 per cent solution. When perfusion of the normal left side was attempted, the animal stiffened, gasped, took a deep breath and ceased to breathe. In spite of prolonged and continuous artificial respiratory measures the animal would not breathe. In about ten minutes the lower extremities quivered and clonic contractions of the lower and upper extremities were noted. It was the impression of the operator at the time that he had possibly gone through the inferior or posterior wall of the sinus into the subarachnoid space or more likely that the major portion of the perfused solution had gone down the posterior nasal ostia into the lungs, the cat being fairly deep under ether anesthesia at the time. Careful examination of the skull, however, revealed no perforation of the inner walls of the sinus. The lungs on examination showed marked emphysema peripherally.

Cat 33.—In the second group of cats the perfusion was similarly attempted. This time the anesthesia was purposely made light and the head was held well forward to prevent, if possible, perfusion into the posterior part of the pharynx. A reaction was obtained in this animal identical with that observed in the former instance. Benzyldimethylamine methiodide in a 1 per cent solution was successfully perfused through the infected sinus, but when perfusion of the normal side was attempted this animal also gasped, took a deep breath and would not breathe again in spite of more prolonged artificial respiration. Clonic contractions set in as before. This time regurgitant vomiting occurred five minutes after respiration ceased. Autposy revealed no perforation into the brain cavities and no obstruction of the trachea or the larger bronchi. Marked emphysema was noted in both lungs, especially peripherally.

The lungs were tied at the tracheal junction and fixed immediately in a solution of formaldehyde. Microscopically, sections of the lungs showed marked congestion of the capillaries and blood vessels, with strong contraction of the larger vessels, that is, the pulmonary artery and vein.

No other animal had suffered from a reaction similar to this with any of the other solutions or in the operator's previous experience. In the third group of cats, benzyldimethylamine methiodide in a 1 per cent solution was again perfused through the sinuses of two different animals. In both these instances a response was obtained similar to that obtained with the other drugs. No unusual reaction occurred.[2]

Cat 4-36.—*Right Sinus.*—There was chronic infection of the right sinus. This cat had acute sinusitis for about three and one-half weeks. It was sick during the first week, being lethargic and mean. During the second and third weeks it was more attentive and friendly, but suffered a severe head cold. The sinus was perfused as before with betaphenylethylamine in a 1 per cent solution. On dissection of the membrane it was noted that it was markedly thickened, dull and adherent. A large quantity of purulent material was present within the sinus. It was especially thickened and more adherent at the site of injection. Microscopically, the membrane was greatly thickened, with much fibrosis, and was filled with plasma cells in barricade-like layer rather deep below the epithelium. Between this layer and the epithelium there was a zone containing fewer plasma cells but numerous polymorphonuclears. The epithelium was greatly thickened, showing numerous goblet cells and migrant polymorphonuclears, and the cilia were partly denuded. Some mucous cysts extended into the tunica propria. The epithelium and the underlying zone gave some evidence of acute irritation superimposed on the chronic infection. Part of the membrane was extremely edematous, greatly thickened and completely infiltrated with leukocytes, plasma cells and lymphocytes. The epithelium contained numerous migrant leukocytes, many of which had reached the surface. In patches the epithelium had disappeared, but there was evidence of regeneration through squamous cells.

Left Sinus.—The sinus was perfused with betaphenylethylamine in a 1 per cent solution. In this specimen the membrane was somewhat thickened throughout and was also somewhat dull, but at the site of injection it was much more thickened and somewhat more hyperemic than the usual normal membrane. Microscopically, the membrane was greatly thickened, slightly edematous and hyperemic. The epithelium showed destruction of its surface, with only small patches of its cilia, and these were greatly injured. There was much proliferation of epithelium, as shown by the many layers and numerous mitotic figures.

Cat 11-36.—*Right Sinus.*—Chronic infection was present. This animal belonged to the second group. The sinus was perfused with tetrandrine methiodide in a 1 per cent solution. In this case a second perfusion of the solution was done twenty-four hours after the first, and the animal was killed seventy-two hours after the second injection. The cat was not acutely ill at any time. A nasal discharge, however, was noted for about one week. After the first perfusion the cat revived well and was in apparently good health at the time of the second. The second perfusion was similarly withstood, and the animal was killed on the third day after it. The membrane was not markedly thickened but was somewhat

2. K. K. Chen in a personal communication stated: "Betaphenylethylamine hydrochloride is slightly more toxic than ephedrine sulfate but is decidedly much less toxic than neo-synephrin. All these figures were obtained by intravenous injection."

adherent on dissection. Considerable hyperemia was present. Microscopically, the membrane was greatly thickened, edematous, hyperemic and filled with plasma cells. There were numerous polymorphonuclears and some eosinophils. The epithelium had been entirely destroyed in patches; elsewhere it showed considerable injury; the cells were tall; goblet cells were not conspicuous, and cilia were destroyed or clumped together. There were deep crypts in which the epithelium appeared more nearly normal, except that the goblet cells were more numerous. In these crypts the cilia appeared normal. No sign of processes of repair was present.

Left Sinus.—The uninfected left sinus was perfused twice with tetrandrine methiodide in a 1 per cent solution. In this case the membrane was not thickened but was somewhat adherent. Moderate hyperemia was present at the site of injection. Microscopically, the mucosa was thin but hyperemic. The epithelium was made up of low columnar to squamous-like cells without cilia anywhere and was entirely destroyed in patches. A considerable number of polymorphonuclears was present in the tunica propria, indicating a subacute inflammation. The injury to the epithelium was the most marked feature of this membrane. The sinus cavities were filled with fibrin and leukocytes.

Cat 22-36.—*Right Sinus.*—Chronic infection was present. The sinus was perfused twice in the aforementioned manner with hordenine methiodide in a 1 per cent solution. This cat suffered an acute infection for three weeks. Afterward the infection subsided. The membranes were dull and thick but were freed easily from the long wall. Moderate hyperemia was present. The sinus was filled with purulent material. Microscopically, the membrane was thickened to about five times normal. Plasma cells and leukocytes were scattered in the tunica propria, but were not very numerous. Occasional small patches of lymphocytes were present. The epithelium was tall and pseudostratified, with fairly numerous goblet cells filled with mucus; the cilia were intact, and slight hyperemia was present.

Left Sinus.—The sinus was perfused twice with hordenine methiodide in a 1 per cent solution. The membrane was thicker than a normal membrane but was not adherent. Slight thickening was present at the site of injection, with slight hyperemia at this point. Some mucus-like material was present within the sinus. Microscopically, the membrane was about normal in thickness; the epithelium was intact, consisting of low columnar cells with tall intact cilia; some hyperemia was present, and the lypmhocytes were somewhat more numerous than normal. The cilia were destroyed in patches, and the epithelium showed signs of injury.

Cat 33.—The cat died as already described.

Cat 44-36.—*Right Sinus.*—Chronic infection was present. Two perfusions were made with betaphenylethylamine in a 1 per cent solution. The infection in this cat was moderately acute. A discharge of a purulent nature was present for two weeks following the injection. This cat was sick following the first perfusion and was submitted to the second perfusion with some difficulty. For two days after this operation it was feared that the cat would die, but on the third day it revived considerably and was killed in the usual manner. Dissection of the membrane was difficult because of the marked thickening and apparent friability, the membranes being very adherent to the long walls. Marked hyperemia was grossly present. Microscopically, the membrane was thickened and strongly edematous in the deeper part, with large spaces filled with grayish-staining substance. The tunica propria was somewhat hyperemic, with scattered plasma cells and polymorphonuclears. The epithelium was tall, with numerous goblet cells. The cilia were clumped and destroyed in patches; elsewhere they were fairly numerous.

Left Sinus.—The sinus was normal, and two perfusions were given; the membrane was more dull and thicker than the usual noninfected membrane. It was markedly thickened at the site of injection. It was also more hyperemic than usual and similar to the membrane in which betaphenylethylamine in a 1 per cent solution was used before. Microscopically, the membrane was thin, with low columnar to cubical cells; the cilia were clumped in many places; elsewhere no sign of injury was noted; no hyperemia was present, and there were few cells in the tunica propria.

Cat 111-36.—*Right Sinus.*—Chronic infection was present, and one perfusion with tetrandrine methiodide in a 1 per cent solution was given. The animal suffered a nasal discharge for one week. The membrane was thickened and adherent; a moderate amount of purulent material was present within the sinus. No gross hyperemia was noted. The membrane was thickest at the site of injection.

Left Sinus.—Chronic infection was present, but the sinus was not perfused. Some thickening was present, but it was not as great as that in the usual infected sinus. No hyperemia and little adherence to the wall was noted.

Cat 222-36.—*Right Sinus.*—Chronic infection was present, and two perfusions with hordenine methiodide in a 1 per cent solution were given. A moderately severe infection was present throughout the whole six weeks preceding perfusion. After the first perfusion the animal was sick for one day but revived well from the second perfusion. The membranes in this case were moderately thick and adherent. Slight hyperemia was noted grossly. Microscopically, the mucosa was thickened; considerable fibrosis was present; plasma cells and leukocytes were scattered but not numerous; deep cysts with broken-down epithelium and mucus were present; the surface epithelium was tall and pseudostratified, with partially destroyed cilia.

Left Sinus.—Chronic infection was present; the sinus was not perfused. The membrane was thick and adherent. No hyperemia was noted. Microscopically, the membrane was not greatly thickened; little, if any, sign of infection was present; the epithelium was normal, and the cilia were somewhat clumped together.

Cat 333-36.—*Right Sinus.*—Chronic infection was present, and two perfusions with benzyldimethylamine methiodide in a 1 per cent solution were given. The infection was mild, nasal discharge persisting for four days. The usual reaction was obtained by perfusion. The membrane was moderately adherent and moderately dull and thick. No hyperemia was present grossly. Some hemorrhage was noted at the site of injection. Microscopically, the membrane was thin, the epithelium showing low columnar cells, with numerous goblet cells and marked indications of injury to the cilia.

Left Sinus.—Chronic infection was present, but the sinus was not perfused. The membrane was thickened more than is normal but less than that in the usual chronically infected sinus. Slight thickening, slight adherence to the wall and mild hyperemia were visible throughout. Microscopically, the membrane was somewhat thickened, with considerable hyperemia; the epithelial cells were tall; numerous goblet cells were present, and the cilia were about normal in appearance.

Cat 444-36.—*Right Sinus.*—Chronic infection was present, and one perfusion with a 1 per cent solution of betaphenylethylamine was given. The cat was sick the first two weeks of infection. A profuse nasal discharge persisted for four days. The usual reaction was obtained by perfusion. It had apparently recovered fully at the time of perfusion at six weeks. After the perfusion the cat became sick again and remained semicomatose until it was

killed at the end of the third day. The sinus was filled with purulent exudate, which was thick and adherent. Hyperemia was apparent throughout. Microscopically, the membrane was somewhat thickened, with rather few cells; a few extravascular eosinophils were present in the tunica propria; the epithelial cells were tall, numerous goblet cells being present. The cilia were about normal. Comparatively few cells were present, except that here and there were patches of polymorphonuclears. The blood vessels showed numerous eosinophils. The whole picture was one of slight injury, with little change from the injection.

Left Sinus.—Chronic infection was present, but the sinus was not perfused. This membrane was also hyperemic, thick, dull and moderately adherent. The sinus contained a moderate amount of purulent exudate. Microscopically, a typical picture of chronically infected membrane was present, with numerous plasma cells, edema, very tall epithelial cells and numerous mucus-filled goblet cells. The cilia were somewhat clumped but not greatly injured.

CAT 103-36.—*Right Sinus.*—Chronic infection was present; the sinus was perfused once with benzyldimethylamine methiodide in a 1 per cent solution. This cat suffered a mild infection with a nasal discharge for eleven days. It was in excellent health at all times. The animal was seemingly unaffected by the perfusion or the anesthesia. The membrane was moderately thickened, slightly hyperemic and adherent. Some hemorrhage was apparent, and increased thickening was evident at the site of injection. Microscopically, a typical picture of chronically infected membrane was present, with numerous plasma cells, some edema, tall epithelial cells, the goblet cells not being conspicuous, patches of lymphocytes, and epithelium destroyed in patches, elsewhere the cilia being somewhat clumped but not greatly injured.

Left Sinus.—Chronic infection was present; the sinus was perfused once with tetrandrine methiodide in a 1 per cent solution. The membrane was moderately thickened, slightly adherent and slightly hyperemic. Some thickening was present at the site of injection. Microscopically, there were a greatly thickened membrane, marked edema and considerable hyperemia. The tunica propria was filled with plasma cells and numerous polymorphonuclears. The epithelium was somewhat taller than normal but intact. The cilia showed little sign of injury. Goblet cells were more numerous than usual.

PATIENT 2-H-36.—A middle-aged man had a history of chronic sinusitis for ten years. He had been given chlorbutanol and ephedrine inhalant nose drops and later had received numerous irrigations of the antrum over a period of about six months. He complained of headaches, frequent colds and pain over the left antrum. There was roentgenographic evidence of marked involvement of the sinus. On irrigation, a considerable quantity of exudate was found in the left antrum. Two cubic centimeters of hordenine methiodide in a 1 per cent solution was injected into the sinus and left there. Forty-eight hours later the process was repeated. A smaller amount of purulent exudate was present. Two cubic centimeters of the solution was again injected. The patient, quizzed six hours after each injection, claimed that there was no increase or decrease of symptoms. The process was repeated forty-eight hours later. This time a small amount of mucopurulent material was present. The patient reported no change in his condition. Twenty-four hours after the final injection the sinus membrane was removed by the Caldwell-Luc operation. The membranes were somewhat adherent, but the sinus wall was removed intact. It was markedly thickened and fibrous. No purulent exudate was present within. Microscopically, the membrane was greatly thickened; the deep part was edematous and had lymph follicles, and the more

superficial part was infiltrated with lymphocytes and plasma cells and had every indication of an old chronic infected membrane. Lymph follicles were found beneath the epithelium, which was denuded in patches and elsewhere greatly injured, showing only the basal layer of cells with the more superficial columnar cells in various stages of destruction. Occasionally small patches of columnar cells with nearly normal cilia were found. There were numerous folds of epithelium below the general surface, some forming cryptlike recesses. In these the epithelium was about normal save for some infiltration of leukocytes. Occasional plasma cells showed two nuclei. There was some indication of an increased number of polymorphonuclears, with some migration of polymorphonuclears through the epithelium.

PATIENT 4-36.—A middle-aged man had a history of acute exacerbations of chronic sinusitis for at least five years. He suffered from marked pain below the left eye and a persistent headache in the left frontal region.

Like the former patient, this man was given three irrigations of the antrum with sterile water; these were followed by injections of 2 cc. of betaphenylethylamine in a 1 per cent solution. This patient, however, reported marked benefit and relief from pain following the injections; in fact, he was on the verge of refusing operation after the third injection. As with patient 2 the purulent exudate was much reduced at the time of the third irrigation. The membrane was thick, adherent and fibrous. Several small polyps were found within the cavity, but no purulent exudate was present. Microscopically, the membrane was greatly thickened and very edematous, with vascular congestion. The membrane was infiltrated with plasma cells. Much of the epithelium was denuded, with only the basal layer of cells remaining. Here and there even the basal cells were gone, exposing the tunica propria to the surface. Elsewhere the epithelium was present and of the stratified columnar type but showed various degrees of destruction. Beneath the epithelial layer were numerous cysts of various sizes.

CONDUCTION DEAFNESS

STATISTICAL OBSERVATIONS

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Usually the impairment of hearing by air conduction in patients with conduction deafness is accounted for readily by either lesions of the middle ear or otosclerotic lesions. When such patients have impairment of hearing by bone conduction the customary explanation is that there is also a lesion of the inner ear, the cochlear nerve or the auditory pathways of the central nervous system, i. e., that the patient has a so-called combined deafness. For some patients this is doubtless the correct explanation. However, the available evidence gives but poor support to the view that it is always the correct explanation.

The present investigation reports the incidence of impairment of hearing by bone conduction in a group of 516 patients who with either one or both ears heard better by bone than by air conduction. Furthermore, the present study seeks to determine by statistical analyses of the clinical histories whether or not the differences in acuity of hearing by bone conduction are related to any of the following items: age, appearance of the tympanic membrane, tinnitus, familial deafness and the degree of impairment of hearing by air conduction.

MATERIAL AND METHOD

The material analyzed consists of the clinical histories of patients examined in this laboratory. Of the examinations made during the six year period from September 1928 to August 1934, all histories are utilized that satisfy the following requirements: (1) that for the Weber, Schwabach and Rinne tests a tuning fork of the frequency of 512 double vibrations per second was used; (2) that the tuning fork was heard longer by bone conduction than by air conduction (negative Rinne test); (3) that for those patients with unilateral impairment of hearing there was definite lateralization of the sound to the affected ear in the Weber test; (4) that the Western Electric Company 1-A audiometer was used in making the audiogram; (5) that the responses of the patient were, in the opinion of the examiner, reliable, and (6) that the tests were made under satisfactory conditions as to room noise, etc.

Of the 516 patients whose records satisfy these requirements, 136, or 26 per cent, had unilateral impairment, and 380, or 74 per cent, had bilateral impairment. Since the two ears of a person with bilateral impairment of hearing often have different thresholds for both air and bone conduction and differ also with respect to other clinical signs, each ear is considered separately. The total number of ears included in this study, therefore, is 896.

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There is no entirely satisfactory method of grouping this material with respect to the acuity of hearing by bone conduction, even though all the bone conduction tests utilized in this study were made with tuning forks of the same frequency (512 double vibrations). The classification adopted is based on the length of time the tuning fork was heard by bone conduction by the patient as compared to the examiner. This method is more reliable than would be a classification based on the actual number of seconds the tuning fork was heard by each of the patients. The latter method is not satisfactory because (1) tuning forks, even of the same frequency, differ greatly from each other in the length of time that they can be heard normally by bone conduction, (2) the rate of damping of any tuning fork is affected by the method of holding and of using the fork during the test and (3) it is impossible to make proper corrections for the masking effect of extraneous sounds on the auditory thresholds of the patients not examined in the sound-proof room (about 25 per cent of the group).

The terms used to designate the three groups into which the patients are divided with respect to acuity of hearing by bone conduction are the usual ones: normal, prolonged and shortened. However, the limits for inclusion in the group with normal bone conduction are much wider than is customary. Therefore, in order to avoid confusion in the minds of those who read the paper, the three terms are placed in quotation marks whenever used. In the group of patients with "shortened" bone conduction are included only those who heard the tuning fork not more than half as long as did the examiner.[1] Therefore in the "normal" group are included not only those patients who heard the fork as long as did the examiner, but also those who are customarily classified as having a "slightly" or a "moderately" shortened hearing by bone conduction. By this classification, "prolonged" bone conduction means only that the patient heard the tuning fork longer than did the examiner under the conditions of the test.

The reliability of this method of classification is of course dependent on a knowledge of the examiner's acuity of hearing by bone conduction. All the examiners who have participated in the testing of any of these patients were less than 40 years of age, and all had good hearing. Some, however, heard slightly better by bone conduction than did others. In grouping the material allowances have been made for these known differences between examiners.

It must be emphasized that this classification is an arbitrary triple and unequal division of the distribution of a continuous variable. Pohlman and Kranz,[2] Knudsen and Jones,[3] Hallpike[4] and others have shown by careful experiments that environmental noise, by reducing the acuity of hearing by bone conduction of the examiner, exaggerates the relative acuity of hearing by bone conduction of the patient whose hearing by air conduction is impaired. However, these experiments are not sufficiently comprehensive to warrant the conclusion that a "prolonged" bone conduction time is always due to this factor. It is a common clinical observation that certain changes in the middle ear apparently produce a condition favoring the transmission of sound by bone conduction. Further, it is an undeniable

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1. A more comprehensive discussion of the basis for this definition of "shortened" bone conduction time may be found in the papers by Ciocco and Weinstein (Am. J. M. Sc. **187**:100, 1934) and by Ciocco (Acta oto-laryng. **22**:529, 1935).

2. Pohlman, A. G., and Kranz, F. W.: Ann. Otol., Rhin. & Laryng. **35**:113, 1926; **39**:927, 1930.

3. Knudsen, V. O., and Jones, I. H.: Bone Conduction, Arch. Otolaryng. **13**:489 (April) 1930.

4. Hallpike, C. S.: J. Laryng. & Otol. **45**:1, 1930.

fact that even in a sound-proof room most patients with unilateral disease of the middle ear lateralize the sound of the fork in a Weber test to the diseased ear. Unless one believes that a unilateral lesion of the middle ear affects the inner ear of the other side, it must be concluded that with some lesions of the middle ear there is a better transmission of sound by bone conduction. A relative lengthening of bone conduction time occurs in cases of unilateral impairment, and there appears to be no reason why it cannot also take place in cases of bilateral impairment of the sound-conducting apparatus. Finally, the observation that some persons hear better by bone conduction than does the examiner may be regarded as only another expression of the wide range of normal variation common to all carefully studied biologic phenomena; it would therefore be surprising not to find examples of "prolonged" bone conduction time. What has been said concerns relative acuity of hearing by bone conduction. It would be futile to discuss the so-called "absolute" bone conduction time as long as the mechanics of bone conduction itself are unknown.

The distribution of the 896 ears of this study, grouped according to the foregoing definitions, is: "prolonged" bone conduction, 26.7 per cent; "normal" bone conduction, 66.1 per cent; "shortened" bone conduction, 7.2 per cent.

TABLE 1.—*The Relation of Hearing Acuity by Bone Conduction to Age*

Bone Conduction Time	Age in Years	
	Mean	Standard Deviation
"Prolonged"	31.70 ± 0.51	11.59 ± 0.56
"Normal"	36.15 ± 0.35	12.46 ± 0.24
"Shortened"	49.64 ± 1.17	13.93 ± 0.82

RELATION OF HEARING ACUITY BY BONE CONDUCTION TO OTHER FACTS

Age.—The mean ages, classified according to bone conduction time, are given in table 1. The patients with "prolonged" bone conduction time were, on the average, about five years younger than those with "normal" bone conduction time, and these in turn were, on the average, about thirteen years younger than the persons with "shortened" bone conduction time. These differences are statistically significant.

In a previous paper [1] I showed that age is the only clinical factor associated with a disproportionate shortening of the bone conduction time and that, on the average, the older the person the shorter is the bone conduction time when the test is made with a tuning fork of 512 double vibrations. This conclusion is based on a study of 907 patients who had good hearing by air conduction, at least up to 1,024 double vibrations (in 6 cases 512 double vibrations was the highest frequency that was heard well). In my opinion the significance of this finding is that the perception of sound by bone conduction, besides being dependent

on the condition of the cochlear nerve and its end-organ, is related to some other as yet unknown factor or factors which are directly associated with age. The fact that the patients in the present study, all of whom had impaired hearing by air conduction but whose hearing by bone conduction was better than that by air conduction, also showed a similar relation of age to hearing acuity by bone conduction, strengthens the foregoing conclusion

Hearing Acuity by Air Conduction.—The audiograms made for the 896 ears studied have been divided into groups according to the classification of Guild.[5] It should be noted that into each classification are grouped audiograms for ears with a small and well defined range of variation in the acuity of hearing by air conduction. This method of classification takes into account the degrees of impairment for each frequency, and not only for 512 double vibrations, the frequency used for the bone conduction tests.

The incidence of each type of audiogram for ears with "prolonged," "normal" and "shortened" bone conduction time, respectively, is shown in table 2. From this table the following facts are worthy of note: 1. All types of audiograms, from those indicating a relatively slight degree of impairment of hearing, such as the B_1 type, shown in the chart, to those indicating a marked degree, such as the D_4 or B_4 type, are found associated with each of the three groups as to bone conduction time, but with a different incidence. 2. The distribution of the various types of audiograms is practically the same for the groups of patients with "prolonged" and with "normal" bone conduction time; in these two groups the B_1 and B_2 types occur more commonly. 3. The most frequent type of audiogram for those persons with "shortened" bone conduction time is the D_4, and consequently the distribution of the types of audiograms in this group differs markedly from that in the other two groups.

The differences in distribution, shown in table 2, when studied with reference to the age factor, are found to be independent of age. A detailed tabulation is not presented. For each type of audiogram the difference in age for persons with "prolonged," "normal" and shortened" bone conduction time is of the same order as has been noted for the group of patients as a whole.

The evidence is conclusive that, on the average, the acuity of hearing by air conduction is better in those patients with "prolonged" or with "normal" bone conduction time than in those with "shortened" bone conduction time. The similarity in distribution of the types of audiograms in the first two groups is regarded as evidence that prolonged bone conduction time represents, in reality, merely the upper extreme of the

5. Guild, S. R.: A Method of Classifying Audiograms, Laryngoscope **42**: 821, 1932.

normal range of variation. The fact that in the group with "normal" bone conduction time are included all those patients who heard the fork more than half as long as the examiner indicates that the lower extreme of the normal range of variation of bone conduction time is near that arbitrarily chosen for the grouping of this material.

The Appearance of the Tympanic Membrane.—The appearance of the tympanic membrane, as described in the records of the otoscopic

TABLE 2.—*Incidence of Types of Audiograms for Ears with Each Type of Bone Conduction Time*

	Bone Conduction Time					
	"Normal"		"Prolonged"		"Shortened"	
Type of Audiogram	Absolute Number	Percentage	Absolute Number	Percentage	Absolute Number	Percentage
B_1	145	24.5	59	24.7	2	3.1
B_1 $C_{8\text{-}8}$	22	3.7	20	8.4	1	1.5
B_1 $C_{10\text{-}12}$	1	0.2	..	...	3	4.6
B_2	198	33.4	81	33.9	6	9.2
B_2 $C_{8\text{-}8}$	26	4.4	3	1.3	6	9.2
B_3	27	4.6	3	1.3	4	6.2
B_4	6	1.0	..	...	6	9.2
D_1	6	1.0	2	0.8	..	...
D_2	16	2.7	8	3.3	..	...
D_3	45	7.6	19	7.9	2	3.1
D_4	37	6.3	6	2.5	31	47.7
E_1	3	0.5	1	0.4	..	...
E_2	15	2.5	13	5.4	..	...
E_3	7	1.2	3	1.3	1	1.5
E_4	2	0.3	3	1.3	1	1.5
F_1	9	1.5	7	2.9	..	...
F_2	18	3.0	8	3.3	..	...
F_3	9	1.5	3	1.3	2	3.1
Total	592	99.9	239	100.0	65	99.9

TABLE 3.—*Relation of Hearing Acuity by Bone Conduction to the Appearance of the Tympanic Membrane*

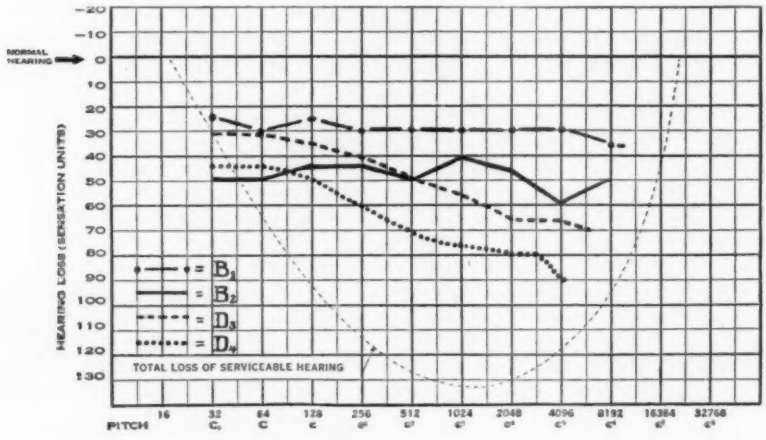
	Bone Conduction Time			
Appearance of Tympanic Membrane	"Prolonged"	"Normal"	"Shortened"	Total
Normal	82	164	16	262
Slight changes	78	217	22	317
Marked changes	41	94	10	145
Perforation	10	44	15	69
Total	211	519	63	793

examinations, has been classified as follows: normal, slight changes (slight to moderate retraction, slight thickening, etc.), marked changes (marked thickening and retraction, extensive calcifications, etc.) and perforations. These conditions are distributed as follows in the ears studied: normal, 33 per cent; slight changes, 40 per cent; marked changes, 18.3 per cent, and perforations, 8.7 per cent.

The relationship between the acuity of hearing by bone conduction and the appearance of the tympanic membrane is given in table 3. The distributions shown in this table are not proportional to the totals, and

the chi-square test clearly indicates that there is a discrepancy between the expected distributions based on chance alone and the observed distribution. This discrepancy is due essentially to the relatively greater number of normal tympanic membranes associated with "prolonged" bone conduction time and to the unexpected higher incidence of perforated tympanic membranes associated with "shortened" bone conduction time.

In table 4 is shown the relationship between the appearance of the tympanic membrane and the hearing acuity by air conduction for the four types of audiometric curves most frequently found in this material. Examples of these four types of audiograms are given in the chart. Analysis by means of the chi-square test shows that the relative dis-



Actual examples of the B_1, B_2, D_3 and D_4 types of audiogram.

tributions for the types of audiograms are significantly different. The differences are apparently due to association between (1) the B_1 type of audiogram and marked lesions of the tympanic membrane, (2) the B_2 type and a normal tympanic membrane and (3) the D_4 type and a perforated tympanic membrane. These positive associations are also obtained when to each of the four common types of audiograms are added those similar in general form but differing in some detail.

One definite clinical syndrome, suggested by the different associations between the appearance of the tympanic membrane and the acuity of hearing by air and by bone conduction, is confirmed by a survey of the individual cases. The three items, D_4 type of audiogram, "shortened" bone conduction time and a perforated tympanic membrane (often with an aural cholesteatoma), are frequently found together in cases of severe chronic infections of the middle ear of long duration. It is

noteworthy that, in agreement with classic statements made in textbooks, many patients ascribe their aural infection to an attack of scarlet fever.

Tinnitus.—No association between the complaint of tinnitus and the length of bone conduction time is observed. In the group as a whole, 60.9 per cent of the patients complained of tinnitus. Of those with "prolonged" bone conduction time, 60.9 per cent had this symptom, and it was also found in 60.7 per cent of those with "normal" bone conduction time and in 62.9 per cent of those with "shortened" bone conduction time.

Family History of Deafness.—Data regarding the incidence of hearing impairment in the patient's family are not sufficient for a detailed genetic study. For the purposes of this investigation a "positive" family history means that one or more members of the family had an impairment of hearing which was apparently not due to age, trauma or infection.

TABLE 4.—*Relation of Hearing Acuity by Air Conduction to the Appearance of the Tympanic Membrane.*

Appearance of Tympanic Membrane	Type of Audiogram				Total
	B_1	B_2	D_3	D_4	
Normal	60	93	25	15	193
Slight changes	73	79	20	38	210
Marked changes	58	26	14	7	105
Perforation	16	4	3	10	33
Total	207	202	62	70	541

Satisfactory replies were obtained from 398 patients; of these 176, or 44.2 per cent, gave a history of familial deafness. Of those with "prolonged" bone conduction time, 56.4 per cent gave a positive history; only 44.3 per cent of those with "normal" bone conduction time and 37.8 per cent of those with "shortened" bone conduction time gave a similar history. The differences between the percentages are statistically significant and indicate an association between prolonged bone conduction time and familial deafness. Definite information regarding the types of hearing impairment in the other members of the families of these patients is not available; therefore, conclusions from this association are not warranted.

SUMMARY AND COMMENT

This paper presents data on the acuity of hearing by bone conduction for 516 patients (896 ears) with a conductive type of impairment (acuity by bone conduction being better than that by air conduction). The data have been statistically analyzed to determine whether or not the acuity of hearing by bone conduction is related to any of the fol-

lowing items: age, the appearance of the tympanic membrane, tinnitus, a history of familial deafness and the degree of impairment of hearing by air conduction. The findings are as follows:

"Prolonged" bone conduction time, as defined, was found in 26.7 per cent of the cases, "normal" bone conduction time in 66.1 per cent and "shortened" bone conduction time in 7.2 per cent.

There is a statistically significant age difference between the persons of the three groups. Those with "prolonged" bone conduction time were, on the average, the youngest; those with "shortened" bone conduction time were the oldest.

On the average, the acuity of hearing by air conduction of persons with "prolonged" and with "normal" bone conduction time was similar and was more acute than in patients with "shortened" bone conduction time.

A normal tympanic membrane was observed with greater relative frequency in persons with "prolonged" bone conduction time, while a perforated tympanic membrane was found relatively more often in those with "shortened" bone conduction time.

The incidence of tinnitus was practically the same for each group.

The incidence of a family history of deafness was highest in those with "prolonged" bone conduction time and lowest in those with "shortened" bone conduction time. The differences are statistically significant.

The effect of age on the acuity of hearing by bone conduction appears to be the most noteworthy finding. Prolonged bone conduction time in all probability represents the upper "tail" of the frequency distribution of the normal variation. The fact that the persons with such bone conduction were the youngest, on the average, may be regarded as confirming the hypothesis that acuity of hearing by bone conduction is associated with some factor or factors other than the condition of cochlear nerve and end-organ.

A METHOD FOR THE EARLY DETECTION OF OTOSCLEROSIS

A STUDY OF SOUNDS WELL ABOVE THRESHOLD

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This is a preliminary report of some experiments undertaken to find a method for detecting threatened or potential ankylosis of the stapes before there is any loss of hearing as shown by the usual functional tests. The approach was by way of sounds louder than those of minimum audibility. The idea is to diagnose clinical otosclerosis (or other dyscrasia of the bone about the windows of the labyrinth) so early that experimental observations and preventive treatment may be instituted at a time when they will be of most service. There is no evidence that anything can be done to reverse the otosclerotic process, but it does stop at various stages of development, and early detection would open up possibilities for treatment now unavailable. The background for my experiments was laid on the following observations and hypothesis.

OBSERVATIONS

Throughout the ages acuity of hearing has been measured at minimum audibilty (by air conduction and by bone conduction), but sounds of this intensity are rarely listened to except under special circumstances. They do not necessarily give a true picture of acuity of hearing for louder sounds. The ear does not usually listen to sounds of faint intensity. It ignores them. For ordinary conversation the normal ear expects and is accustomed to sounds from 40 to 60 decibels above normal minimum audibility.

Many persons do not appear to hear loud sounds as well by air conduction or by bone conduction as would be expected from their threshold audiograms. This is one reason why some do not hear as well with hearing aids as is expected. It suggests that there may be lesions which interfere proportionately less with faint sounds than with louder sounds.

Many other persons appear to hear much better than their threshold audiograms imply, and loud sounds disturb some markedly deafened persons as much as they do those with good hearing. This suggests that there may be lesions which interfere proportionately more with faint sounds than with loud sounds—lesions which even in some deafened ears permit near normal sensitivity for loud sounds.

My data obtained by masking often show discrepancies between different ears with the same threshold sensitivity. This is in line with the preceding observations.

Deafness from otosclerosis often appears suddenly, in one ear before the other, and many months or even years may intervene before the ears show equal deafness. Before a certain date there has been no difficulty in hearing; afterward there is a definite and usually a progressive loss. This suggests a critical incident in the growth of the lesion, such as the moment of contact or coalescence of calcified areas invading the annular ligament. Thereafter there would be a definite limitation of motion, but the stapes would be immobilized only when and mainly where the annular ligament was completely bridged by new bone.

Measurements of both the minimum audible intensity and the above-threshold intensity in normal and subnormal ears are necessary to investigate these phenomena and to establish a better understanding of how the ear hears. Hearing by both air conduction and bone conduction should be measured.

To measure the sensation of loudness it is necessary to use the patient's ears as reference, because there is no other way to determine whether or not the two ears hear exactly alike.

To measure the sensation of loudness produced by sounds above threshold intensity by air conduction it has been proposed to use a reference tone of say 1,000 cycles at 70 decibels above the normal threshold and to balance this against other frequencies in the same ear.[1] With untrained observers the result obtained by this method is not as satisfactory as desired, because large differences in frequency make comparative values for loudness uncertain, and small differences measure too nearly the same thing. There are also other reasons, which I shall not discuss at this time.

HYPOTHESIS

If the annular ligament is but slightly involved by an otosclerotic process, that is, some time before there is bony ankylosis of the stapes, very small excursions of the stapes (those at or near minimum audibility) would not be appreciably affected (being extremely microscopic) but if the excursions are slowly increased (as by louder sounds) a point will be reached at which freedom of excursion will be definitely and suddenly limited (hobbled) by the narrowed or shortened annular ligament. (In other words, the extent of excursion will then be less than normal.)

1. MacKenzie, Donald: The Relative Sensitivity of the Ear at Different Levels of Loudness, Physical Rev. **20**:331, 1922. Kingsbury, B. A.: Direct Comparison of the Loudness of Pure Tones, ibid. **29**:373 and 588, 1927. Steinberg, J. C.: The Function of the Apical Turns of the Cochlea and the Symptoms of a Lesion in this Location: IV. From the Point of View of the Physicist, Tr. Am. Otol. Soc. **25**:131, 1935.

No matter where about the circumference of the annular ligament narrowing occurs, it will act to (hobble) limit excursion of the footplate at and beyond a critical amplitude of movement. The threshold of hearing might be normal, but sounds above a critical intensity would be sensed below the normal expectancy. The threshold would then not be a dependable criterion for sounds louder than this critical intensity. This effect would be best detectable at low frequencies, because here the movement of the stapes is slower and of greater excursion and therefore more easily measurable (there are other reasons). The physicists and physiologists are best fitted to work out mathematically the exact formulas of performance. Otologists are interested more in methods and in diagnosis, treatment and preventive implications.

METHODS

Eighteen or twenty ways were devised to measure the critical amplitude. The most obvious was, beginning at minimum audibility, to raise the intensity in small decibel steps or, beginning near the upper limits of serviceable intensity, to diminish the intensity in small steps. When the critical intensity is reached, the ear should not be able to detect as small a minimum audible change of intensity (increase or decrease) as is expected at such intensity or as is detectable at the other points of the intensity scale. Various modifications of this simple method were tried, such as slow and fast alternating tones and beating tones. I found evidence of diminished discrimination at various points in the intensity scale in certain persons with normal and with abnormal hearing.

The next method of approach was by masking methods, that is, by determining the loudness by air conduction (in decibels) required to mask sounds of different intensity applied by bone conduction to the same ear (the opposite ear being completely masked) and vice versa. The idea is to detect the hobble by the increase necessary to mask a given intensity over that required in normal ears or in opposite ears. Various modifications were tried out with short and long, and slow and fast, intermittent and beating tones.

These last methods, though theoretically possible, proved difficult for untrained observers. Most of them were difficult even for trained subjects, but I am still experimenting with them.

All efforts were directed toward comparing what an ear heard with what it would be expected to hear, but the habit of measuring hearing and masking at or near minimum audibility was so strong that it was some time before I thought of another and simple way to determine, both in apparently normal ears and in subnormal ears, whether or not an ear hears sounds at various intensities above threshold better or

worse than would be expected from threshold audiograms and whether or not there is a boosting or a restraining effect at certain intensities in certain ears.

The method is to have the patient compare sounds of known loudness thrown alternately into his ears (to compare intensities binaurally). This is accomplished by the patient indicating in which ear the sound appears the louder, or whether it appears of equal loudness to the two ears. The examiner varies the loudness in the worse ear until it appears equal to that heard in the better ear (or vice versa); in other words the sensation of loudness is made the same in both ears; they are balanced. I call this the "alternate binaural loudness balance." [2] The method is simple, and the data are surprisingly accurate even with untrained observers (including little children). It is assumed that at each frequency the better hearing ear is preferable for reference. This may not always be true.

If the intensities necessary for the obtaining of binaural balance are different for the two ears, the spread, or difference in intensity, is measurable, being the additional number of decibels required to produce the same sensation of loudness in the worse ear over the number required for the better ear (at a given intensity)—in other words, the added weight in sound pressure necessary to balance or equalize the sound in the two ears. With bone conduction or combined air and bone conduction the technic of elicitation is different, but the principle is the same.

The experiments have revealed four distinct phenomena.

1. In certain persons there is a constant difference (or no difference) in the loudness necessary for binaural balance at all intensities.

2. In certain persons there is a changing difference in the loudness necessary for binaural balance at different intensities; that is, a poorer ear may appear to hear loud sounds disproportionately well as compared to faint sounds. (There is a greater increment of loudness with similar changes in intensity above threshold than in the better ear.)

3. A combination effect of 1 and 2 may exist.

4. Anomalies in discrimination of intensity occur; that is, in certain ears at critical intensities, small changes in loudness appear greater or less than they do to the average normal ear or greater or less than to the opposite ear.

I have reduced the phenomena to quantitative measurement.

I show but one graph to illustrate each of four distinct groups of lesions and phenomena. I have many similar graphs. Some two hun-

2. In other words, the alternate binaural loudness required to balance.

dred patients have been tested by the methods described. The method of graphing is not wholly satisfactory.

In addition to demonstrating the results obtained by means of the "alternate binaural loudness balance" the graphs suggest several practical applications and possibilities in addition to those which I originally had in mind. There are fascinating possibilities for experimentation with animals in the physiology of hearing and especially in differential diagnosis between intracranial lesions and those in the auditory cortex, nerves, ganglions and end-organs. They may furnish an explanation of some of the oddities of bone conduction and of the apparently peculiar results of experiments on animals with sounds of 70 decibels or other loud tones. The Weaver and Bray phenomena lend themselves well to such investigations.

Human ears are able to judge bilateral differences in loudness accurately, possibly because of the necessity and habit of using binaural hearing for locating distant sources of sound. It may be that the binaural balance method will prove of more value as a means for detecting the hobble effect than monaural tests. Only time will tell.

In children and adults with normal or subnormal hearing who had family histories of otosclerosis, what appears to be the hobble effect has been found in a few ears, and in different ears at various intensity levels. The accumulation of convincing data will be slow. The reverse of the hobble (the boosting effect) has been found in many deafened ears, especially in association with what is called nerve deafness. Neither the hobble, or "impedance," effect or the reverse, the boosting, helping or aiding effect, was found in clinically established uncomplicated otosclerosis.

The alluring thought is that maybe all this means that this is a test not only for detecting the hobble in an ear showing no loss of hearing at threshold but for a deafened ear as well and possibly a test for estimating the degree of involvement from the sclerosing or hobbling process. This may also be a method for aiding in differential diagnosis of deafness caused by neural involvement and by lesions of the middle ear. It may mean nothing of the kind. The method has already proved useful in overcoming uncertain observations caused by such factors as inattention, nervousness and tinnitus, because sounds 10 or 15 decibels above threshold are so loud that they are definitely heard, easily balanced binaurally and not masked or confused by the tinnitus.[3] It is hoped that the method will point a way to a better understanding of auditory function.

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3. No matter what minimum audibility indicates, sounds 10 or 15 decibels above the threshold will mask the tinnitus and will be loud enough to be easily heard and balanced binaurally. The balance will then show the relative sensitivity of the two ears at these intensities.

Figure 1 *A* shows audiograms of the right and left ears of a patient with bilateral obstructive deafness. The audiogram of the right ear was obtained both by air conduction and by bone conduction while the better left ear was masked. The bilateral loudness balance (or difference) is shown by the length and angle of slant of the diagonal lines drawn across the ordinates at frequencies 128, 256 and 1,024. The uppermost diagonal line joins the levels of threshold audibility of the right and the left ear. At 128 there is a difference of 15 decibels between the sounds necessary to produce the same sensation of loudness in the two ears. That is, it is necessary to put into the right ear 15 decibels more than it is necessary to put into the left ear to obtain a bilateral balance of loudness. This is true at all intensities, but for clearness it is indicated only at threshold and at two other intensities in the graph. At 256 there is a difference of 20 decibels between the sounds necessary to produce a sensation of the same degree of loudness in the two ears. At 1,024 there is a difference of 30 decibels between the sounds necessary to produce the same sensation of loudness in the two ears.

The method of plotting was devised so that it could be used on the patient's audiogram chart. From a clinical standpoint it is satisfactory, as it not only shows at a glance the binaural balance (or difference) of loudness under each frequency tested but gives a picture of the relative loudness balance or unbalance as between the frequencies without additional charting facilities. It is not entirely satisfactory from the physicist's standpoint, and I therefore show in figure 1 *B* another method of plotting.

Figure 1 *B* is a graph plotting the binaural balance of loudness in the same ears and for the same three frequencies as those in figure 1 *A*. For each frequency the lower left plot is at the threshold of audibility for the frequency indicated.

The levels of loudness are plotted along the ordinates for the right ear and along the abscissas for the left ear. Each plotted point indicates the number of decibels above the normal thresholds which produced sensations of equal loudness in the two ears. The line making a 45 degree angle with the ordinates and the abscissas indicates where the points would be if the two ears heard exactly alike.

In these two ears the plotted points for the frequencies 128, 256 and 1,024 make straight lines parallel to the 45 degree line. That is, they show no variation in the loudness balance throughout the range of intensity. In other words, these ears hear sounds louder than threshold in exactly the same ratio to each other as they heard those of threshold intensities. The level of intensity required for the right, or worse, ear is above that required of the same sound heard in the left,

or better, ear. This difference varies with frequency. The exact amount in each case is indicated by the distances of the plotted points from the point of equal balance on the 45 degree line.

When the two sounds appear equally loud, the difference in the levels of intensity required for balancing indicates the loss of hearing in the worse ear as compared to that in the better ear. In these two ears there is no variation of the relative loss of hearing at any of the audible intensities. This is the universal supposition on which all functional tests have been heretofore predicated. It will be shown to be an erroneous supposition in certain types of deafness.

This graph also shows the hearing of one ear plotted in terms of that of the other.

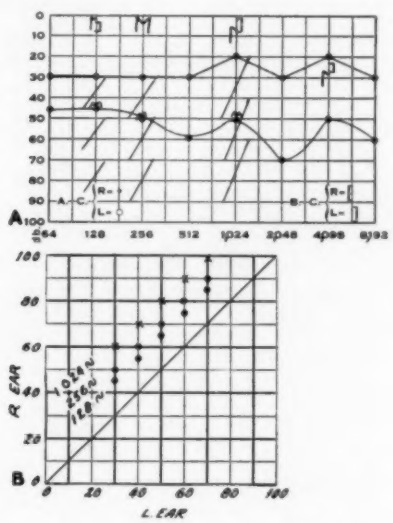

Fig. 1.—*A*, audiograms of the right and left ears of a patient with bilateral obstructive deafness. *B*, graphs showing the binaural loudness balance in the same ears and for the same frequencies shown in *A*.

Figure 2 shows audiograms of a patient with monaural obstructive deafness. It shows a greater spread, or difference, between the intensities necessary to obtain a balance in loudness than is shown in figure 1 *A*, because one ear is normal and the other is markedly deafened. The slope and length of the diagonal lines at all four frequencies—128, 256, 1,024 and 4,098—are generally greater than those shown in figure 1 *A*, but nearly all are parallel at each frequency.

When the hearing of one ear is plotted against that of the other as shown in the graph in the left lower corner, the plottings at all frequencies make a straight line, except for a slight tendency of 256

and 1,024 to approach the line of equal balance at intensities over 80 decibels in the right ear. The ease of obtaining definite graphs should be noted. With two exceptions in over one hundred cases of pure conductive, obstructive or mechanical lesions, I found a strong tendency to an undisturbed bilateral loudness balance throughout the intensity scale.

Bone conduction was normal for both ears at all frequencies, and air conduction for the worse ear was down to near the 50 decibel line, as in figure 1 *A*. In these two ears there was little or no variation in the relative loss of hearing of the right and the left ear at any intensity, for the frequencies 256 and 1,024. This, as in figure 1 *A*, is what would be expected if measurements made at threshold are believed to show the complete picture of hearing acuity. At a frequency of

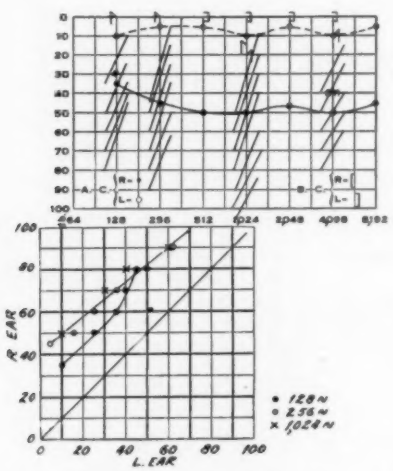

Fig. 2.—Audiograms and graphs showing the binaural loudness balance of a patient with monaural obstructive deafness.

128 there is a tendency for a greater spread, or unbalance, as loudness is increased. This effect is found more often with low tones. If it occurs only at some critical intensity and not thereafter, it is indicative of a hobble effect.

Figure 3 shows the binaural loudness balance in an instance of unilateral nerve deafness. Hearing by bone conduction coincides with that by air conduction in both ears. The diagonal lines indicate a gradual and progressive transition from a marked difference between the intensities necessary to produce a sensation of equal loudness in the two ears to practically no difference above 90 decibels over normal minimum audibility.

The graph below the audiogram shows a plot of one ear against the other and clearly demonstrates this change from marked difference to no difference for two frequencies (256 and 1,024). This tendency to equalize the loudness necessary for balance has been noted in all instances of unilateral nerve deafness. It is not what would be expected if the measurements of the threshold are taken as a complete picture of acuity of hearing. It shows that for sounds well above its threshold a markedly deafened ear may hear almost if not quite as well as a normal ear. It shows why to some persons who are hard of hearing loud sounds appear as loud and annoying as to those with normal hearing. It shows one reason why some deafened persons often complain of one's speaking too loudly. If one spoke to this patient at an intensity

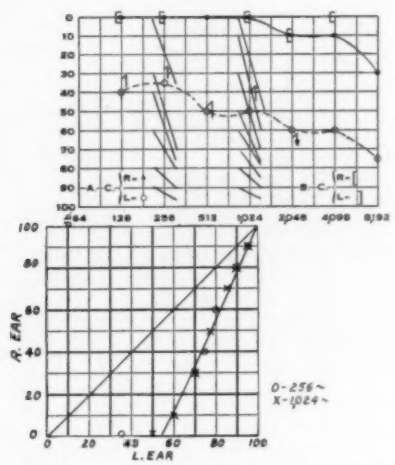

Fig. 3.—Audiograms and graphs showing the binaural loudness balance of a patient with unilateral nerve deafness.

of 75 decibels above normal threshold in the poor ear, the loudness of the voice would appear about as great as that of a 60 decibel sound in his perfectly hearing ear. In other words, if one raised his voice only 15 decibels above the usual level for conversation, the sound would appear almost as loud as that expected by persons with normal hearing, and louder sounds than this would naturally tend to annoy him. The regularity of the occurrence of unexpected recovery in the sensation of loudness with increasing intensity in the poor ear is without question of importance in understanding how the ear functions.

Figure 4 shows the binaural loudness balance in a case of mixed nerve and obstructive deafness. The hearing by bone conduction for both the right and the left ear is about the same as that by air conduc-

tion except for the higher frequencies, at which the hearing by bone conduction appears to be better than that by air conduction in each ear. The lines showing binaural balance indicate a gradual and increasing tendency to become horizontal with increasing loudness (except at fre-

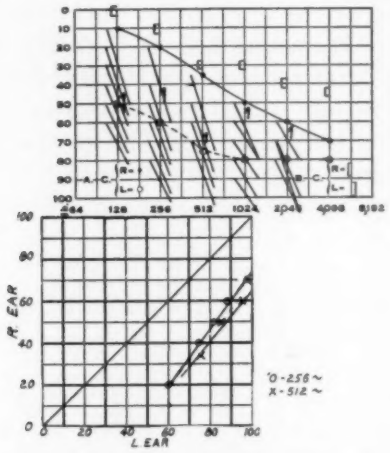

Fig. 4.—Audiograms and graphs showing the binaural loudness balance in a case of mixed nerve and obstructive deafness.

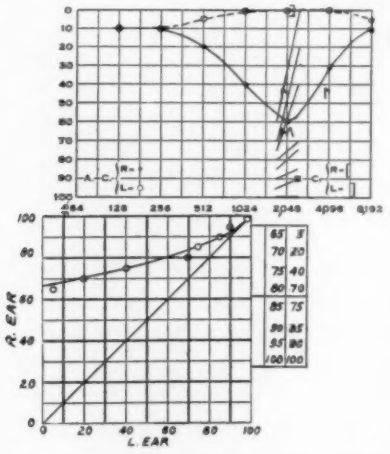

Fig. 5.—Audiograms of a patient with monaural deafness with a dip in the curve for one ear.

quency 2,048, where the thresholds are close). This is also shown for frequencies 256 and 512 in the graph below the audiogram. The lines joining the plots are not quite parallel with the 45 degree line of the graph. They tend to approach a parallel but do not reach it even for

the louder sounds. This indicates a combination effect. It shows that the observations made with air conduction and bone conduction at threshold do not give a complete picture of the way these ears hear sounds above the threshold intensities.

There is not sufficient recovery or boost in the deafer ear to produce a perfect binaural balance of like intensities, such as is shown in figure 3, but such a tendency is apparent at all lower frequencies.

Figure 5 shows the audiograms of a patient with monaural deafness with a dip in the curve for one ear (right), most marked at 2,048. The hearing by bone conduction nearly coincides with that by air conduction for the poor ear, and there is no deafness evident in either ear at very low or at very high frequencies. From a spread of 60 decibels at threshold, the binaural loudness balance (or difference) lessens, until at 100 decibels the sound appears to be equally loud to both ears. This means that in the right ear it was necessary to increase the intensity only 40 decibels, whereas in the left, or normal, ear it was necessary to increase the intensity 100 decibels to obtain a balance in the two ears with sounds of equal loudness. Both the figures and the graph below the audiogram show clearly this phenomenon of bilateral equalization of loudness with increasing intensity. In other words, as the sounds become louder, they sound more and more alike to the two ears. This substantiates the findings shown in figures 3 and 4 in an ear with a marked dip in the curve for hearing.

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EFFECT OF ZINC IONIZATION AND GALVANIC CURRENT ON THE REACTION OF THE NASAL MUCOSA TO VASOMOTOR DRUGS

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Intranasal ionization with zinc sulfate is attended by destructive changes in the nasal mucosa (McMahon,[1] Hollender and Gorin [2] and Smit [3]). These changes consist of coagulative necrosis and desquamation of surface epithelium, atrophy and loss of glands, extravasation of red blood cells, leukocytic infiltration, fibrosis of the submucosa and thickening of the periosteum. Unless a chronic inflammatory process becomes established, the denuded epithelium is replaced by stratified dedifferentiated epithelial cells, which give rise to normal ciliated columnar epithelium within from six weeks to three months. During this time secretion is diminished because of the loss of functional secreting cells of the intra-epithelial and subepithelial glands. The mucosa presents a dry, shrunken appearance.

The shrunken, blanched appearance of the nasal mucosa, as observed clinically and experimentally, may be the result of several changes induced by ionization. Koeppen [4] showed that a galvanic current of 24 milliamperes may produce complete paralysis of the vasomotor nerve endings and thrombi of the smaller vessels and capillaries. One of us [5] showed that zinc iontophoresis stops the circulation of blood in the web of the frog's foot and in the external ear of the rabbit in from forty to sixty minutes. Clumping of the red blood cells was

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2. Hollender, A. R., and Gorin, M.: Influence of Ionization on Vasomotor Rhinitis: Clinical and Experimental Studies, Illinois M. J. **68**:493, 1935.

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observed in the frog within fifteen minutes. Galvanic currents of the same intensities as those employed for zinc ionization (0.8 to 1.5 milliamperes) did not produce these changes. It is possible that the increase in connective tissue following zinc ionization interferes with the blood supply to the affected part, but this would be a later rather than an immediate change.

The turgescence of the mucous membrane and rhinorrhea in a considerable percentage of persons with vasomotor and allergic rhinitis clear up after intranasal ionization. This relief from symptoms may persist for many months after the lining epithelium has returned to the normal ciliated columnar type. In seeking an explanation for this altered reaction two possibilities were thought of: 1. Changes in the epithelial lining may affect the rate of absorption of substances introduced into the nose. It is a well established fact that practically all accessible mucous membranes have good powers of absorption. This ability to absorb varies under certain conditions. A mucous membrane with a denuded epithelium and a good blood supply absorbs readily. A membrane with an epithelial lining of the stratified squamous type, such as appears during the stages of regeneration following zinc ionization, may be rendered less permeable and made to offer greater protection to the underlying structures against irritating substances. 2. Disturbances of the vasomotor mechanism may be brought about by the process of ionization. The theory adhered to by Eppinger and Hess,[6] Cooke,[7] Hajos[8] and others, that the manifestations of allergy are due in part to an imbalanced autonomic nervous system or vagotonia, has had considerable support. The symptomatic relief produced by epinephrine and other sympathomimetic drugs lends further support to this theory. However, it can be readily seen that any agent that would paralyze or interfere with the vasodilator mechanism would produce symptomatic relief without necessarily altering the state of sensitivity (Alexander[9]). In order to put these views to a test the experiments reported here were devised.

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PROCEDURE

Two groups of healthy adult cats were prepared by intraperitoneal injections of sodium amytal solution. In one group the left side of the nose was filled with 2 per cent zinc sulfate in 10 per cent tragacanth gel. A fine zinc electrode covered with cotton and moistened with 2 per cent zinc sulfate solution was then inserted. In the other group the left side of the nose was filled with 0.85 per cent sodium chloride in 10 per cent tragacanth. A platinum electrode wrapped with cotton soaked in physiologic solution of sodium chloride was inserted. The negative

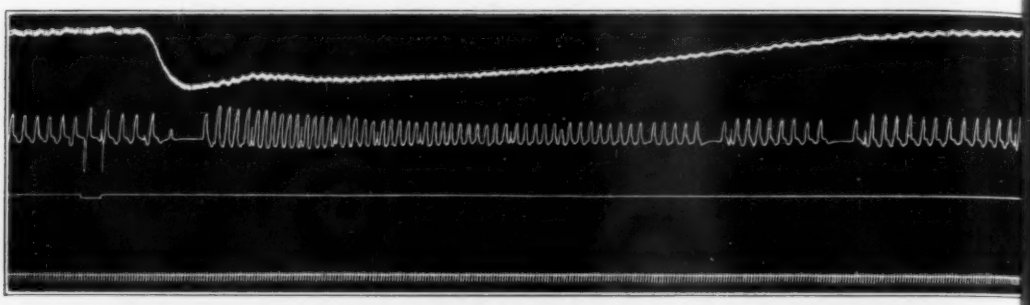

Fig. 1 (cat 15).—Tracing taken one month after zinc ionization of the left side of the nose. The record shows a fall in blood pressure following the instillation of 0.25 cc. of a 1 : 500 solution of mecholyl into the right nostril.

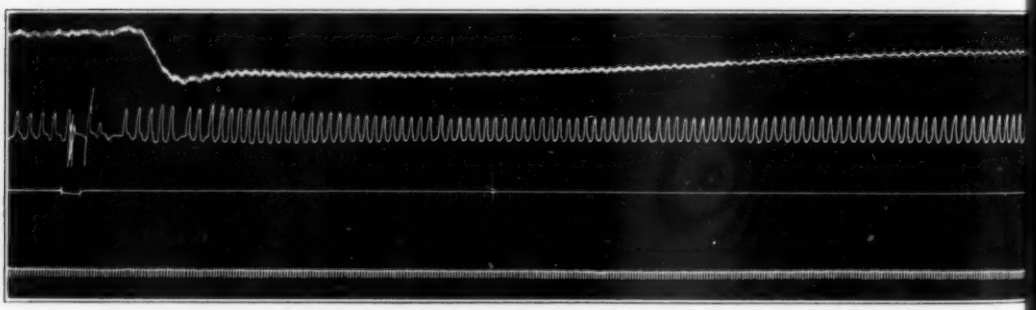

Fig. 2 (cat 15).—Fall in blood pressure after the instillation of 0.25 cc. of a 1 : 500 solution of mecholyl into the left nostril.

electrodes wrapped with cotton soaked in a solution of sodium chloride were placed on the abdomen. A galvanic current of 4 milliamperes, supplied by a "B" battery, was allowed to flow for twenty minutes.

At intervals varying from one week to three months following ionization with zinc sulfate or anodal galvanism, the animals were again placed under sodium amytal anesthesia, and tracheal and carotid cannulas were inserted and connected with a tambour and mercury manometer for recording respiration and blood pressure in the carotid artery. The choanae were then blocked by postnasal cotton plugs saturated with petrolatum. The vasoconstricting or vasodilating agents in amounts of 0.25 to 0.5 cc. were then instilled into either the right or the left nostril,

and after the blood pressure had returned to the normal level an equal amount of the same solution was placed in the other nostril. At the end of the experiment the animal was killed and the nose was placed immediately in a 10 per cent dilution of solution of formaldehyde U. S. P. and prepared for histologic study.

Epinephrine hydrochloride in a 1:100 solution and a 10 per cent solution of neo-synephrin hydrochloride were the vasoconstricting drugs used. These concentrations were selected because of the fact that weaker solutions when placed in the

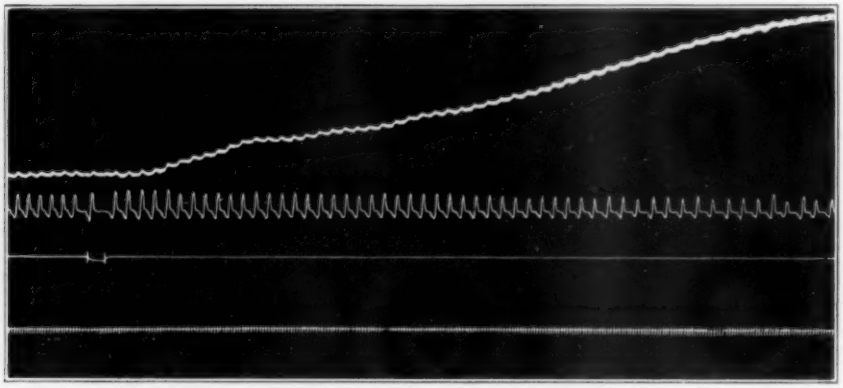

Fig. 3 (cat 19).—Tracing taken three weeks after zinc ionization of the left side of the nose, showing rise in blood pressure following the instillation of 0.25 cc. of a 10 per cent solution of neosynephrin hydrochloride into the left nostril.

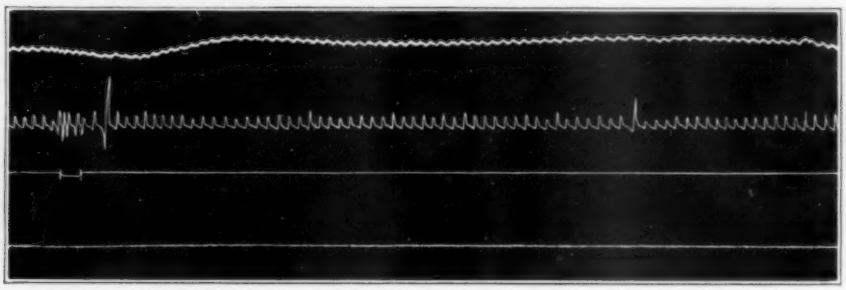

Fig. 4 (cat 19).—Tracing showing a rise in blood pressure after the instillation of 0.25 cc. of 10 per cent solution of neosynephrin hydrochloride into the right nostril.

nose are ineffective in producing any noticeable change in blood pressure. Mecholyl (acetylbetamethylcholine chloride) in a 1:500 solution was selected as the vasodilating agent.

The results of the experiments are given in tables 1 to 4. The time of onset and the extent of the fall in blood pressure produced by mecholyl when placed in the ionized and nonionized sides of the nose showed no significant differences. With epinephrine and neo-synephrin

TABLE 1.—*Effect of Zinc Ionization on Rate of Absorption and Reaction of Nasal Mucosa to Mecholyl*

Cat Number	Time Following Treatment	First Instillation	Fall in Blood Pressure; Time of Onset and Duration; Right Nostril	Fall in Blood Pressure; Time of Onset and Duration; Left Nostril	Histologic Changes in Nasal Mucosa
2	1 wk.	Both nostrils; 1:500 mecholyl	.....	.....	Capillaries and blood spaces on right side greatly dilated
3	2 wk.	Right nostril	10 mm. of mercury in 53 sec.; began in 30 sec.; duration 3 min., 56 sec.	7 mm. of mercury in 52 sec.; began in 33 sec.; duration 2 min.; 52 sec.	Dilatation of vascular structures on right side
4	2 wk.	Left nostril	19 mm. of mercury in 45 sec.; began in 4 sec.; duration 9 min.	16 mm. of mercury in 45 sec.; began in 10 sec.; duration 20 min.	Right side greatly congested
5	3 wk.	Left nostril	11 mm. of mercury in 56 sec.; began in 24 sec.; duration 13 min., 40 sec.	16 mm. of mercury in 46 sec.; began in 28 sec.; duration 43 min.	Right side greatly congested
15	1 mo.	Right nostril	23 mm. of mercury in 40 sec.; began in 22 sec.; duration 54 sec.	20 mm. of mercury in 45 sec.; began in 31 sec.; duration 10 min., 38 sec.	Slight difference in dilatation; pus present both sides
13	6 wk.	Right nostril	20 mm. of mercury in 55 sec.; began in 31 sec.; duration 6 min., 40 sec.	18 mm. of mercury in 46 sec.; began in 25 sec.; duration 4 min., 15 sec.	Two sides show equal degree of vasodilatation
14	3 mo.	Right nostril	10 mm. of mercury in 45 sec.; began in 35 sec.; duration 6 min., 10 sec.	11 mm. of mercury in 55 sec.; began in 32 sec.; duration 6 min., 15 sec.	Vasodilatation both sides; pus present both sides

TABLE 2.—*Effect of Galvanic Current on Rate of Absorption and Reaction of Nasal Mucosa to Mecholyl*

Cat Number	Time Following Treatment	First Instillation	Fall in Blood Pressure; Time of Onset and Duration; Right Nostril	Fall in Blood Pressure; Time of Onset and Duration; Left Nostril	Histologic Changes in Nasal Mucosa
22	2 wk.	Right nostril	16 mm. of mercury in 39 sec.; began in 26 sec.; duration 8 min., 5 sec.	10 mm. of mercury in 39 sec.; began in 27 sec.; duration 5 min., 45 sec.	Engorgement of erectile tissue right side
25	1 mo.	Left nostril	15 mm. of mercury in 40 sec.; began in 16 sec.; duration 17 min., 10 sec.	16 mm. of mercury in 40 sec.; began in 19 sec.; duration 21 min., 57 sec.	Greater dilatation of capillaries on right side
26	6 wk.	Right nostril	14 mm. of mercury in 39 sec.; began in 20 sec.; duration 7 min., 10 sec.	9 mm. of mercury in 45 sec.; began in 25 sec.; blood pressure did not return to normal	Same degree vasodilatation both sides
10	3 mo.	Left nostril	13 mm. of mercury in 36 sec.; began in 29 sec.; duration 11 min., 10 sec.	15 mm. of mercury in 39 sec.; began in 25 sec.; duration 16 min.	Greatly engorged blood spaces and dilated vessels both sides

TABLE 3.—*Effect of Galvanic Current on Rate of Absorption and Reaction of Nasal Mucosa to Neosynephrin Hydrochloride*

Cat Number	Time Following Treatment	First Instillation	Rise in Blood Pressure; Time of Onset and Duration; Right Nostril	Rise in Blood Pressure; Time of Onset and Duration; Left Nostril	Histologic Changes in Nasal Mucosa
24	1 mo.	Left nostril; neosynephrin hydrochloride 1:100	No rise	32 mm. of mercury; duration, 1 hr.	Right greatly shrunken; blood vessels constricted
8	1 mo.	Right nostril; neosynephrin hydrochloride 1:100	9 mm. of mercury in 2 min.; began in 20 sec.; duration 6 min., 45 sec.	10 mm. of mercury in 2 min., 25 sec.; began immediately; duration 1 hr.	Right side shows vasoconstriction
27	6 wk.	Right nostril; neosynephrin hydrochloride 1:100	30 mm. of mercury in 1 hr.; began in 42 sec.; duration 1 hr.	No rise	Little evidence of vasoconstriction on either side; both sides infected
11	3 mo.	Left nostril; neosynephrin hydrochloride 1:100	5 mm. of mercury in 4 min., 35 sec.; began in 52 sec.; duration 15 min.	40 mm. of mercury in 9 min., 30 sec.; began in 15 sec.; duration 1 hr.	Same degree of vasoconstriction on both sides

TABLE 4.—*Effect of Zinc Ionization on Rate of Absorption and Reaction of Mucosa to Epinephrine or Neosynephrin Hydrochloride*

Cat Number	Time Following Treatment	First Instillation	Rise in Blood Pressure; Time of Onset and Duration; Right Nostril	Rise in Blood Pressure; Time of Onset and Duration; Left Nostril	Histologic Changes in Nasal Mucosa
7	2 wk.	Right nostril; 10 per cent neosynephrin	2 mm. of mercury in 80 sec.; began in 60 sec.; duration 110 sec.	35 mm. of mercury in 3 min., 40 sec.; began in 105 sec.; death	Sections were not prepared
18	2 wk.	Left nostril; 1:100 epinephrine	13 mm. of mercury in 10 min., 40 sec.; began in 36 sec.; duration 15 min., 15 sec.	35 mm. of mercury in 2 min., 58 sec.; began in 18 sec.; duration 38 min., 50 sec.	Blood vessels markedly constricted on right side only
19	3 wk.	Left nostril; 10 per cent neosynephrin hydrochloride	7 mm. of mercury in 71 sec.; began in 25 sec.; duration 1 hr.	66 mm. of mercury in 5 min.; began in 25 sec.; duration 1 hr.	Vessels not constricted on either side
20	1 mo.	Right nostril; 10 per cent neosynephrin hydrochloride	19 mm. of mercury; duration 56 min.	10 mm. of mercury in 7 min.; duration 1 hr.	Vasoconstriction on right side only
27	6 wk.	Right nostril; 1:100 epinephrine	11 mm. of mercury in 72 sec.; began in 25 sec.; duration 45 min.	No rise	Vessels constricted on both sides
12	3 mo.	Right nostril; 10 per cent neosynephrin hydrochloride	40 mm. of mercury in 17 min., 40 sec.; began in 30 sec.; duration 1 hr.	4 mm. of mercury in 30 sec.; began immediately	Left side not shrunken; pus present on both sides
9	3 mo.	Left nostril; 1:100 epinephrine	32 mm. of mercury in 6 min., 40 sec.; began in 54 sec.; blood pressure did not return to normal level	No rise	Right side shows vasoconstriction; pus on both sides

EXPLANATION OF FIGURE 5

Fig. 5.—*A* (cat 18), the left side of the nose was subjected to zinc iontophoresis two weeks previous to the instillation of 0.25 cc. of a 1 : 100 solution of epinephrine hydrochloride. The section was taken from the superior surface of the nasal cavity and shows flattened epithelium, round cell infiltration and a slight increase in connective tissue; the arterioles, capillaries and venous spaces are not constricted. *B* (cat 18), section of mucosa from the superior surface of the right nasal cavity. The vascular elements show marked constriction. *C* (cat 24), one month after the left side of the nose was treated with the galvanic current alone. A section through the cavernous tissue at the base of the nasal septum on the left side after the instillation of a 10 per cent solution of neosynephrin hydrochloride shows no evidence of vasoconstriction or collapse of the blood spaces. *D* (cat 24), a section through the cavernous tissue of the septum on the right side, showing constricted blood vessels and collapsed cavernous spaces. *E* (cat 3), two weeks following zinc iontophoresis of the left side of the nose. A section through the cavernous tissue of the left side following the instillation of a 1 : 500 solution of mecholyl shows no evidence of vasodilatation or engorgement of the venous spaces. *F* (cat 3), section through the erectile tissue of the septum on the right side after the instillation of mecholyl, showing vasodilatation and engorgement of the blood spaces. *G* (cat 4), two weeks after the left side of the nose was subjected to zinc ionization, mecholyl was instilled. A section through the erectile tissue of the turbinate on the left side shows no dilated blood vessels and no engorged blood spaces. *H* (cat 4), section through the erectile tissue of the turbinate from the nonionized side after treatment with mecholyl, showing dilated blood vessels and greatly engorged blood spaces.

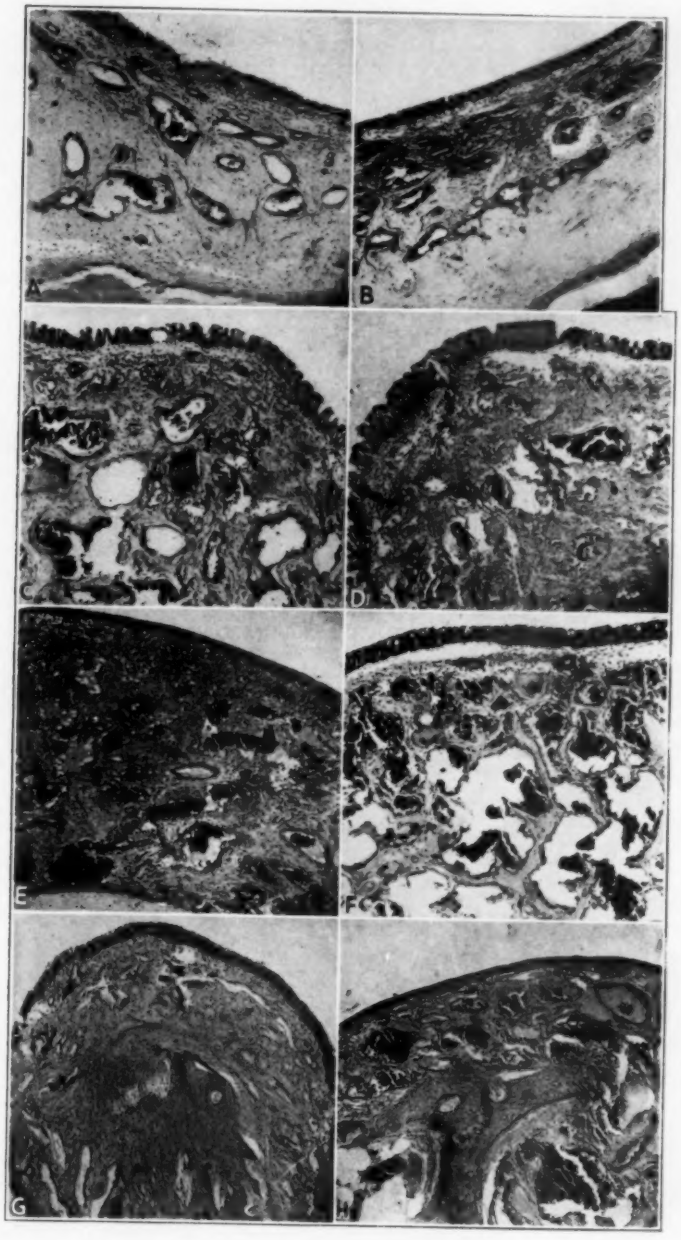

Figure 5

EXPLANATION OF FIGURE 6

Fig. 6.—*A* (cat 10), section through the erectile tissue of the superior surface of the left side of the nose three months after treatment with a galvanic current. The instillation of a 1 : 500 solution of mecholyl produced vasodilatation and engorgement of the venous spaces. *B* (cat 10), section through the cavernous tissue of the right superior surface of the nose after the instillation of a 1 : 500 solution of mecholyl, showing the same degree of vasodilatation and engorgement of blood spaces as on the opposite side. *C* (cat 14), three months after zinc ionization. The cavernous tissue of the left nasal septum shows vasodilatation and congestion following the instillation of a 1 : 500 solution of mecholyl. *D* (cat 14), the cavernous tissue of the right nasal septum shows vasodilatation and engorgement of blood spaces after treatment with a 1 : 500 solution of mecholyl. *E* (cat 2), one week after zinc ionization. A section from the bone of the turbinate, showing a large number of osteoclasts. *F* (cat 17), six weeks after zinc ionization. Synechia between the septum and the turbinate. *G* (cat 12), three months after zinc ionization. A section from the synechia between the septum and the turbinate, showing new bone formation and thickened periosteum. *H* (cat 25), oil immersion photomicrograph, showing eosinophil leukocytes in a capillary after the application of mecholyl to the nasal mucosa.

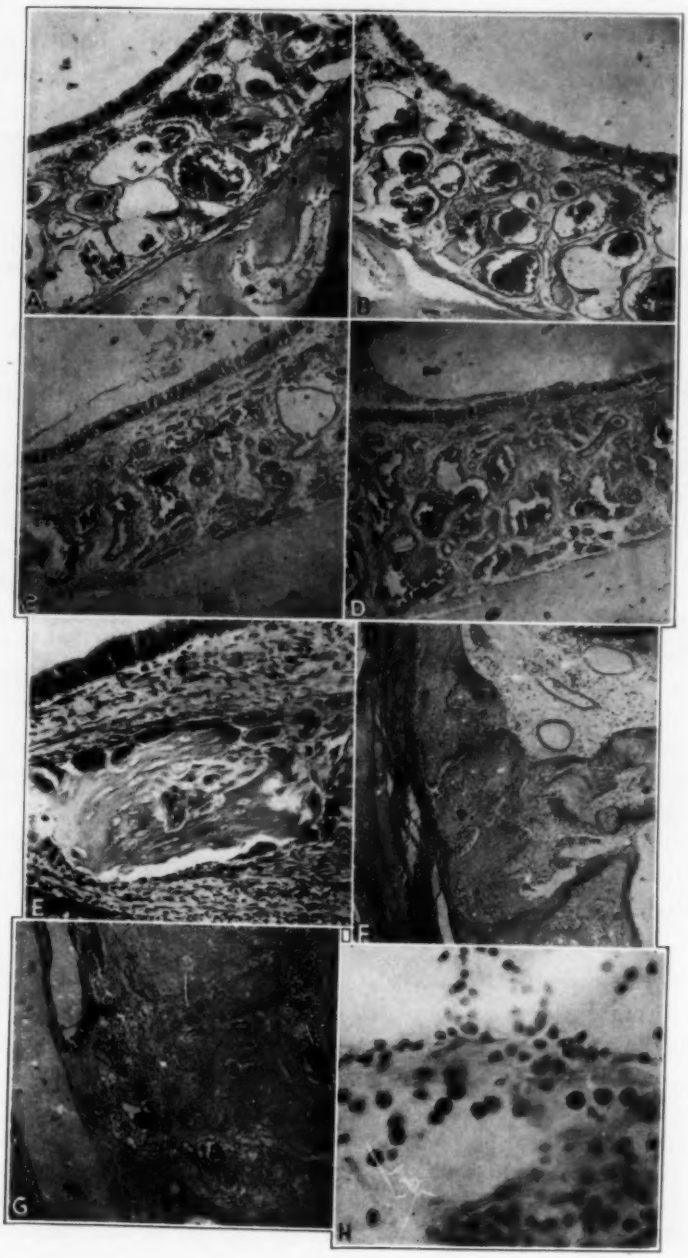

Figure 6

hydrochloride a rise in blood pressure occurred only with the first instillation; the second instillation into the other side of the nose produced no significant rise in blood pressure except in animals 7 and 9. After comparison of the results obtained in an animal in which the first instillation was made into the left nostril with those obtained in an animal in which the first instillation was made into the right nostril, no definite differences could be made out. The results obtained with these vasoconstrictor drugs were not consistent.

Histologic examination of the nasal mucous membranes showed that up to six weeks the zinc-ionized membranes failed to respond to either vasodilator or vasoconstrictor drugs. The nonionized nasal mucosa reacted to these drugs regardless of which side of the nose was selected for the first instillation. The vascular reactions of the ionized and nonionized mucous membranes to these drugs were the same three months after ionization.

The vascular response to vasoconstrictor and vasodilator drugs of the mucosa of those cats subjected to the galvanic current alone was not elicited until four weeks after treatment. In these animals the vasomotor mechanism was similarly disturbed but returned apparently in a much shorter time. In both the membranes ionized with zinc and those treated with galvanic current the presence of infection interfered with the vascular response of these tissues to the drugs used.

The pathologic changes observed in the zinc-ionized membranes were identical with those already mentioned and reported by other investigators. In addition to these changes there was found great osteoclastic activity in the turbinates of cats 2 and 4 one and two weeks after zinc ionization. Synechiae were found in cats 13, 17 and 19. In all membranes treated with the parasympathomimetic drug mecholyl, there was a marked increase in eosinophilic leukocytes in the submucosa.

SUMMARY AND CONCLUSIONS

Changes in the nasal mucosa following zinc ionization do not hinder the absorption of vasomotor drugs instilled into the nasal cavity.

The blood vessels and the cavernous tissue of the nasal mucosa fail to react to vasoconstrictor and vasodilator substances for six weeks after zinc ionization and four weeks after treatment with the galvanic current. This failure of the vascular structures to constrict or to dilate is due either to a temporary paralysis of the vasomotor nerves or to a loss of vascular tone. Histologic study of the blood vessels, however, showed no thickening or other pathologic change in the walls of these structures that might account for this temporary disturbance of the vasomotor mechanism.

THE STAPHYLOCOCCUS IN RELATION TO SINUSITIS, BRONCHITIS AND BRONCHIECTASIS

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There has been almost universal disregard of the staphylococcus as a potential pathogen in the nose, pharynx and bronchi, in spite of the high percentage of staphylococci reported in the numerous bacteriologic studies made of these regions.

Attention has been so focused on streptococci, pneumococci, influenza bacilli, fungi and spirochetal organisms that the staphylococcus has come to be regarded as a common, nonpathogenic inhabitant of these areas and its presence ignored.

Cultures of material from the spheno-ethmoid region of sixty-four patients with chronic low grade sinus infection revealed that hemolytic staphylococci were found forty-eight times (an incidence of 75 per cent), nonhemolytic staphylococci forty-six times (an incidence of 72 per cent), Streptococcus viridans once, hemolytic streptococci once, nonhemolytic streptococci four times, type II pneumococci once, type IV pneumococci five times, Bacillus influenzae three times, Micrococcus catarrhalis four times and, among nonpathogenic bacteria, Bacillus xerosis ten times, Bacillus Hofmanni eleven times, Bacillus aerogenes once and chromogenic micrococci three times.

The cultures were taken after shrinking the nasal mucosa and then carefully wiping a small swab from the vault of the nasopharynx forward over the face of the sphenoid and up into the posterior ethmoid region. The swab was then carefully withdrawn. While there may be some criticism of this technic, I feel that when it is carefully done in routine work its results are sufficiently accurate.

The results of these cultures furnish enough evidence to justify workers in this field in taking a renewed interest in the study of this organism, its clinical significance and treatment along more specific lines.

I should like this discussion to be regarded in the nature of a preliminary report, or rather as a summary of clinical impressions, for I do not feel that I have yet gathered enough data to make a more detailed report or to warrant positive statements.

BACTERIOLOGY

Staphylococci should probably now be classified only as hemolytic and nonhemolytic, because there is abundant evidence that the aureus,

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albus, citreus, and roseus types are merely cultural variations in pigment production. Likewise, there is probably only one exotoxin, which presents various biologic effects, and not hemolytic, leukocytic, necrotizing and lethal toxins, as was formerly supposed.

This toxin is comparable with the toxins of diphtheria and tetanus and is capable of producing rapid death in rabbits, even when injected in minute amounts. However, the ability of various strains to produce this toxin varies widely. Its potency can be determined in the laboratory by its necrotizing action on the rabbit's skin, and it can be converted to toxoid for active immunization. It also produces antitoxin in horses capable of neutralizing the toxin of all strains of staphylococci, so one has at one's disposal for clinical use both toxoid and antitoxin.

The antitoxin content of the patient's serum can also be determined by hemolytic methods and expressed as a titer of so many units according to an international standard.

As a result of three hundred and forty consecutive estimations, the average titer was found to be about 3.20 units per cubic centimeter and the normal range from approximately 1.60 units to 6.40 units per cubic centimeter. In a few instances the titer was as low as 0.10 unit, and in one patient, recovering from osteomyelitis, it was as high as 410 units per cubic centimeter.

In my experience the most marked clinical improvement has occurred in patients treated with toxoid whose titers were below 1.60 units; but definite clinical improvement has also occurred in those with much higher titers. While the amount of circulating antitoxin can be readily increased by giving toxoid, a relatively slight increase is often accompanied by clinical improvement manifested after the first few injections; so I agree with Dolman,[1] who has stated that recovery from staphylococcic infection depends to some degree on the establishment of a certain critical concentration of antitoxic antibodies in the blood, which in some instances need not be much greater than that already present.

It is interesting to note that this rise in titer so readily obtained by the injection of toxoid, which contains both the bacterial body antigen and bacterial toxin, cannot be obtained by the injection of a staphylococcus vaccine.

From the foregoing data, I do not feel that the presence of staphylococci can be ignored, especially when the varied biologic effects of its toxin are considered; and although the cultures may show a nonhemolytic strain, I am not sure that this strain may not be hemolytic at another time, under different conditions. Repeated cultures, with repeated estimations of the potency of this toxin, on the same patient will be necessary before this point can be settled.

1. Dolman, C. E.: Treatment of Localized Staphylococcic Infections with Staphylococcus Toxoid, J. A. M. A. **100**:1007-1010 (April 1) 1933.

The relationship of these facts to acute exacerbations of chronic sinusitis and to the common cold is interesting, for although a marked increase in the number of pathogenic organisms of all kinds is found at this time, the rôle of bacteria and viruses in the production of these conditions is far from being understood.

ACUTE SINUSITIS

When the predominant organism in acute sinusitis is the staphylococcus, there is need for far more concern as to possible complications that may occur than when the organism that is present is the streptococcus. Fortunately, the streptococcus is usually predominant in acute sinusitis, and although there may be grave complications with the more virulent strains, the normal body defenses are sufficient to control them in the majority of instances.

When the staphylococcus is predominant, experience has shown that orbital abscess, osteomyelitis, thrombosis of the cavernous sinus, meningitis, abscess of the brain and septicemia are likely possibilities. For this reason early recognition by bacteriologic smears and cultures is very important, because in addition to the usual procedures used to establish aeration and drainage, treatment along specific lines can be started with the toxoid, with the hope of preventing complications or of better preparing the normal body defenses for them should they occur.

In the presence of toxemia, antitoxin serum can be administered for its neutralizing effect in addition to the antigenic stimulus derived from toxoid. I have also found that when transfusion is considered it is helpful to choose as the donor the one whose blood has the highest antitoxin titer for the staphylococcus. Repeated determinations of the titer of the blood of the patient during the course of the infection is of value from a prognostic point of view.

Treatment along this specific line has been of real service during the past two years, and I feel that I can truthfully state that in some patients these complications have been prevented and that other patients, in whom complications had developed, have been definitely benefited.

CHRONIC SINUSITIS

I have been particularly interested in the study of the group of patients, all too familiar to otolaryngologists, who complain of a postnasal discharge, a stuffy nose, perhaps headache, neuralgic pain, asthenopia and frequently associated sore throat, cough and expectoration. The diagnosis of chronic hyperplastic sinusitis best fits this group of chronic low grade sinus infections, so often associated with chronic pharyngitis or laryngitis and especially with chronic bronchitis and

bronchiectasis. The multiplicity of symptoms is familiar to all, as well as the lack of definite clinical findings and of any specific line of treatment.

The lack of proper aeration and drainage of the sinuses is no doubt the underlying factor in the causation of this condition; it may result from mechanical obstruction, allergy or some particularly virulent and destructive infection. No treatment along specific lines is of much avail unless this underlying factor is eliminated and normal physiologic function restored, as nearly as possible. Each case is a problem unto itself and calls for the keenest judgment on the part of the surgeon, as to whether he should use colloidal silver packs or a constructive or a destructive surgical procedure, the operations ranging from resection of the middle turbinate to a complete pansinus operation. However, this discussion is concerned not so much with surgical treatment as with offering an additional line of attack along specific lines.

When one considers the frequency of infection with the staphylococcus in the spheno-ethmoid region and the fact that injections of toxoid yield clinical improvement associated with a rise in the antitoxin titer of the blood for this organism, it seems justifiable to attribute this condition to a specific type of infection.

CHRONIC SINUSITIS WITH CHRONIC BRONCHITIS AND BRONCHIECTASIS

The finding of a pure growth of staphylococci in a few cases of early bronchiectasis in cultures of endobronchial material, with the same findings in cultures of material from the spheno-ethmoid region, I feel is of some significance.

I am aware of the finding of streptococci and a host of other organisms in cases of bronchiectasis of long standing, but it seems probable that they are secondary invaders.

Since the association of chronic sinusitis with chronic nonspecific infection of the lung is well recognized, a discussion of it is unnecessary here. Likewise, a discussion of the many factors and theories advanced as to the production of bronchiectasis is unnecessary, except that I should like to restate that the pathologic picture of fragmentation and degeneration of muscular and elastic tissue with dilatation of bronchi can be caused by the necrotizing action of this toxin when combined with intrapulmonary pressure.

Many other observations have been made which support this view, and of course there are also many other observations which are not so easy to correlate. It is entirely too early for positive statements, but I feel that future work along this line should be done in order to determine whether or not this organism plays an important etiologic rôle.

In my limited experience, I have felt that specific treatment with toxoid in addition to bronchoscopy and the elimination of sinus disease has been of definite value.

SUMMARY

The frequency of infection with the staphylococcus in sinusitis and early bronchiectasis is noted, and treatment along specific lines with staphylococcus toxoid and antitoxin is advocated, in addition to the older, well recognized methods.

A brief review of the bacteriology of the staphylococcus is given.

Future work along this line, both in the research laboratory and in clinical practice, is urged.

The biologic preparations of the Lederle laboratories have been used throughout this investigation.

TREATMENT OF HEMORRHAGE IN NONHEMOPHILIC PATIENTS WITH AN ESTROGENIC SUBSTANCE

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The primary investigations of Carrol Birch,[1] of Chicago, in 1931 led to the use of ovarian extract in the treatment of hemophilia. Birch reasoned that since the disease occurs only in the male there is something lacking in the male organism which is present in the transmitting female which prevents the development of clinical manifestations of the disease. Working on this theory, she has treated hemophilia with ovarian extract with excellent results, and the use of this in the hands of many others since that time has proved the value of her work.

It was also shown that the addition of ovarian substance lowers the coagulation time of blood from a hemophiliac in vitro.

At the suggestion of A. J. Carlson, Birch demonstrated that when the urine from normal men is concentrated and injected in sufficient quantities into castrated female rats an estrus cycle is produced. The urine of five hemophiliacs failed to do this.

To those who operate on the ear, nose and throat there comes a time when in spite of all preliminary precautions hemorrhage occurs. On surgical traumatization of the tissues, some persons with normal clotting time and no history of hemophilia or apparent reason will acquire oozing surfaces, which are no end of trouble and often a source of anxiety. It is entirely possible that these cases in which there are a normal coagulation time and a normal number of platelets may cloud the picture because of the method of determining the clotting time. Mills, of Tampa, Fla., in a recent discussion of the work of Spoto,[2] of the same city, stated that no coagulation test is of any value unless the blood is taken from the vein and that all his hemophilic patients showed normal clotting time for blood taken from the finger by the usual puncture method, while the venous blood taken at the same time required as long as four hours to clot. He suggested that the clotting time, especially of boys, be determined for blood from the vein prior

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Read before the Escambia County Medical Society.

1. Birch, Carroll L.: Hemophilia, J. A. M. A. **99**:1566-1572 (Nov. 15) 1932; Hemophilia and the Female Sex Hormone: Preliminary Report, ibid. **97**:244 (July 25) 1931.

2. Spoto, J. S.: Treatment of Hemophilia with Ovarian Extract: Report of Two Cases, J. Florida M. A. **20**:9 (July) 1933.

to operation. It must be borne in mind that all bleeders are not hemophiliacs. For this reason I have contended for some time that the puncture method is of no avail, as mixture of tissue juices with the expressed blood alters the clotting time, and on several occasions I have withdrawn blood directly from the vein for observation of clotting time.

The method of operation of estrogenic substance on the elements of the blood has been an interesting item of speculation.

Howell's [3] theory of the clotting of blood is that in the circulating blood there are fibrinogen, calcium salts, prothrombin and antithrombin. The last-named substance holds the prothrombin in combination and thus prevents its conversion to thrombin. When blood is shed, the disintegration of the platelets furnishes material (thromboplastin) which combines with the antithrombin and liberates prothrombin, which in turn is activated by the calcium salts and acts on the fibrinogen. The actual process of coagulation then involves only three factors—fibrinogen, prothrombin and calcium. These three factors exist in the normal circulating blood but are prevented from reacting by antithrombin. Thus, any interference with the number or the breaking up of the blood platelets to inhibit the liberation of thromboplastin would cause the antithrombin to remain united with the prothrombin and thus, in its cycle, to prevent the clotting of blood. Therefore, estrogen, in my mind, causes the disintegration of blood platelets which are otherwise impervious, thus starting the cycle of coagulation.

It occurred to me that if estrogen can and does work in instances of true hemophilia it should be of some value to those bleeders who cannot, either by history or from laboratory or clinical data, be classed as hemophiliacs. I therefore determined to try it, and the results are worth reporting. Four patients, three males of various ages and one woman, were used, all of whom had postoperative bleeding although the clotting time was normal.

REPORT OF CASES

CASE 1.—C. E. P., a white boy aged 12 years, had a clotting time of three and one-half minutes. The tonsils and adenoids were removed. There was slight oozing from the lower pole of the right tonsillar fossa shortly after operation. Coagulation serum was given, and a retention sponge was inserted in the right fossa. The sponge was removed the same afternoon, and there was no more bleeding. Six days later bleeding started during sleep. Examination revealed that the fossae were dry and that the oozing was coming from the postnasal space, from which the adenoids had been removed. Postnasal and anterior nasal tampons

3. Howell, W. H.: The Rôle of Antithrombin and Thromboplastin (Thromboplastic Substance) in the Coagulation of Blood, Am. J. Physiol. **29**:187, 1911.

were inserted. Oozing continued around the packing for three hours, after which 25 rat units of theelin was injected subcutaneously. Bleeding ceased after thirty-five minutes. The packs were removed one at a time over a period of eight hours, with no bleeding following, and there was no more at any time. The patient then made an uneventful recovery. There was no history of hemophilia in the family, and cuts had always healed readily, with only normal bleeding.

CASE 2.—J. H., a white woman aged 39, underwent a tonsillectomy with local anesthesia. The clotting time was four minutes. General oozing from the fossae occurred alternately for three days. Local and general hemostatics were given. Clots formed in the fossae, and oozing continued underneath. Removal of the clots and the pressure sponge caused cessation for several hours. Morphine and other sedatives were given. After six days of oozing 50 rat units of theelin was given, and bleeding ceased permanently after thirty minutes.

CASE 3.—A J. M., a boy aged 3 years, had a clotting time of three minutes. Slight oozing occurred after the tonsils and adenoids were removed. This patient had severe mitral regurgitation, and every precaution against bleeding and strain was desired. At the first sign of oozing of postnasal origin, a pack was inserted in the nose; oozing continued for two hours. Then 25 rat units of theelin was given, with cessation of bleeding in twenty minutes. The pack was then removed and no more bleeding occurred.

CASE 4.—L. M., a white man of 32, underwent a tonsillectomy. The clotting time was four minutes. Bleeding occurred two hours after operation, in the form of general oozing from the floor of the right fossa. Sutures and sponges sewn in did not help. The pillars were sutured at the base, with no result. Bleeding stopped only when a pressure sponge was held in. There was no point of hemorrhage, but general oozing occurred. The bleeding was continuous when the sponge was removed for ten hours. Fifty rat units of theelin was given. The bleeding ceased after thirty minutes and did not recur.

COMMENT

In all these cases there was an apparently normal clotting time, and the throat was dry when the patient left the operating table. In no instance was there arterial or venous bleeding, but slight capillary oozing occurred. In two coagulation serum was given with no apparent result.

It is worthy of note that two of the patients were approximately at the age of puberty and menopause, respectively, and that endocrine disturbance may have been relieved by theelin, the estrogenic substance from the urine of pregnant women. Birch, in a personal communication, stated:

> It is of interest along this line that in our family histories there is a record of five transmitters of hemophilia who bled to death during the menopause. There were also two transmitters who had severe hemorrhages—one nasal, the other uterine. Both these women were past middle age. The examination of the blood as to the elements of coagulation showed it to be entirely normal.
>
> Your results are entirely in harmony with mine.

CONCLUSIONS

Treatment of hemophilia with ovarian extract was demonstrated by Birch to be of value.

Treatment with estrogenic substance is of value for certain types of nonhemophilic hemorrhage following surgical procedures when there is apparent imperviousness or failure of the platelets to break down and liberate thromboplastin, which unites in turn with antithrombin and allows clotting.

Clinical Notes

ROENTGEN STUDY OF THE SPHENO-ETHMOID SINUSES

FERRIS SMITH, M.D., GRAND RAPIDS, MICH.

I wish to present a method of roentgenographic study of the ethmoid labyrinths which permits examination of the individual cells without superimposed neighboring structures and also projects the sphenoid sinus of the same side into a position for examination. The sphenoid sinus is further studied in a lateral and in an axial view.

The positions usually employed for the examination of the ethmoid labyrinth do not permit a study of the individual cells. The Rhese position has furnished the nearest approach to a satisfactory picture, but the position of the projected

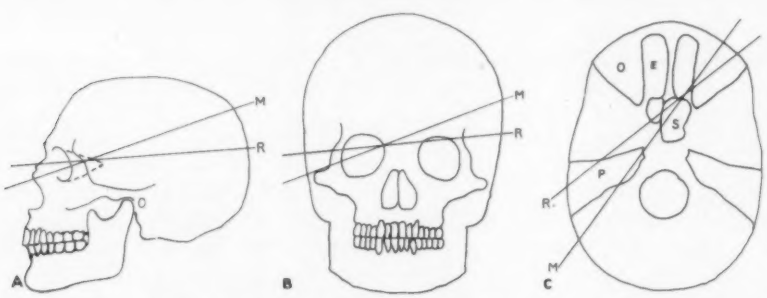

Fig. 1.—In *A* and *B*, the line *R* represents the direction of the ray in the Rhese position. The tube is centered through the orbit from the upper margin of the opposite pinna. *M* indicates the direction of the ray in the modified position. The tube is centered 10 degrees above the line *R*. In *C*, *R* indicates the direction of the ray in the Rhese position and *M*, the direction of the ray in the modified position. This line *M* is 10 degrees mesial to the line *R*. *O* indicates the orbit; *E*, the ethmoid labyrinth; *S*, the sphenoid sinus, and *P*, the petrous portion of the temporal bone.

labyrinth and the superimposed structures frequently render accurate interpretation impossible. This position does, however, offer the best guide to a direction of the rays which will produce the desired result.

It is our desire to project the entire ethmoid labyrinth above the dense line connecting the crista galli and the sella turcica (fig. 2 *B*, *C* and *E*) and at the same time to project the other labyrinth beyond this area. This line represents the crista galli, the cribriform gutter, the roof of the sphenoid sinus and the convexity of the great wing of the sphenoid bone, forming part of the roof of the orbit.

I determine such a position by outlining the entire length of the superior and inferior boundaries of the ethmoid labyrinth and the line from the crista galli to the sella turcica with lead wire and locating a proper angle by trial and error.

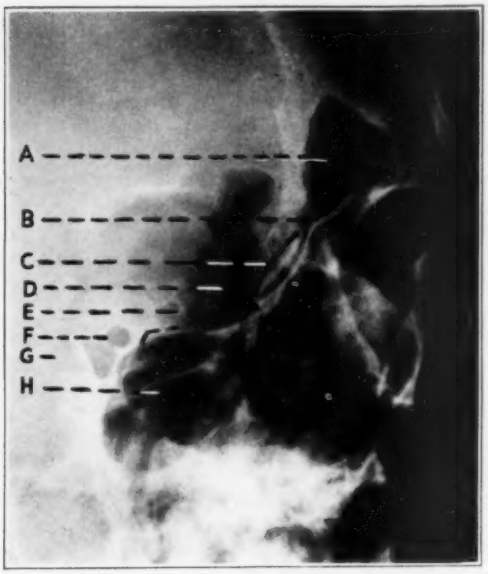

Fig. 2.—The right spheno-ethmoid labyrinth, showing normal sinuses: *A*, frontal sinus; *B*, crista galli; *C*, cribriform gutter; *D*, ethmoid labyrinth; *E*, sphenoid roof; *F*, optic foramen; *G*, orbital margin; *H*, sphenoid sinus. The line indicated by *B*, *C* and *E* runs from the crista galli to the sella turcica.

Fig. 3.—The left spheno-ethmoid labyrinth, showing hyperplasia of the linings of the ethmoid cells and a normal sphenoid sinus.

The technical work was done by Charles Tilson in the service of Dr. T. O. Menees of the Blodgett Memorial Hospital.

The direction of the ray in the Rhese position is through the orbit from the upper margin of the opposite pinna. This is at an angle of approximately 45 degrees to the midplane of the body (fig. 1 *A, B* and *C*). It projects the ethmoid cells into the orbit, with the optic foramen near the outer margin of the orbit. It has the disadvantage of superimposing the ethmoid cells of the two sides.

Our position eliminates this overlapping and projects the entire labyrinth above the dense line running from the crista galli to the sella turcica (fig. 2). In this, the tube is centered above and posterior to the upper margin of the pinna, forming an angle of about 10 degrees in both planes, with the direction of the Rhese position (fig. 1 *A, B* and *C*). The patient's face is placed so that the tip of the nose, the chin and the malar bone are in contact with the cassette.

Figures 2 and 3 are the roentgenograms of the right and the left sphenoethmoid labyrinth of the same patient. The right sinuses are normal; on the left side there is a marked hyperplastic process in the linings of the ethmoid cells and a normal sphenoid sinus.

HYPERPLASTIC LARYNGITIS OF SINGERS

BENJAMIN KATZ, M.D., LOS ANGELES
Laryngologist, Los Angeles County Health Department

When a particular organ is constantly strained during the course of professional work, certain pathologic conditions characteristic of this particular profession develop. In singers incorrect methods of voice training and of singing, the necessity of using the voice during or after colds, neglectful or improper dealing with repeated attacks of sore throat, and inflammation of the larynx usually result in chronic catarrh of the larynx, which sooner or later impairs the functional capacity of the organ. Fortunately, nature created the vocal organ in such a way that the most vital parts of the larynx are resistant and become affected late. Experiments carried out on cadavers demonstrate that if liquid is injected into the mucosa of the larynx it spreads according to certain laws. The liquid accumulates in the interarytenoid space, forming a swelling, and it does not penetrate the true vocal cords. This fact corresponds with the anatomic structure of the larynx, as the mucosa of the interarytenoid space is attached to the underlying structure by loose connective tissue, while on the vocal cords the epithelium is firmly connected to the cord.

When it is exposed to repeated attacks of inflammation, with consequent stasis and swelling due to transudation and proliferation of lymphoid elements in the affected area, the interarytenoid space undergoes a process of hyperplastic enlargement which results in neoformation of connective tissue, and therefore the area becomes permanently swollen. Pathologically this process is identical with cirrhosis of the liver in instances of stasis in that organ. This swelling protrudes into the interarytenoid space like a pad and in the mechanism of the voice serves the same purpose as a mute in stringed instruments, depressing and choking the quality of the sound. At the same time this hyperplastic mass lying between

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Read at a meeting of the Los Angeles Society of Ophthalmology and Otolaryngology, April 27, 1936.

the vocal cords forms a mechanical obstruction and prevents the close contact between the vocal cords which is essential in the production of a clear sound. Besides, this obstruction causes a continuous strain and fatigue of the voice, as the cords must constantly compress this hyperplastic mass in the process of phonation.

CLINICAL PICTURE

The larynx can be of normal appearance, or it may be inflamed if general catarrh is present. The posterior wall of the larynx is swollen and protrudes in the shape of a pad. If catarrh is present, the pad has different shades of red discoloration. In terminal stages, when cirrhosis takes place, pale bluish discoloration appears (fig. 1*A*). As a result of the mechanical obstruction due to the hyperplastic enlargement on the posterior wall, the vocal cords during phonation do not come in close contact in the posterior part of the larynx, and therefore a triangular space of various degree remains permanently open (fig. 1*B*).

PATHOLOGIC CHANGES

In early stages stasis, transudation and proliferation of lymphoid elements are the main histologic features. In later stages neoformation of connective tissue takes place (fig. 2).

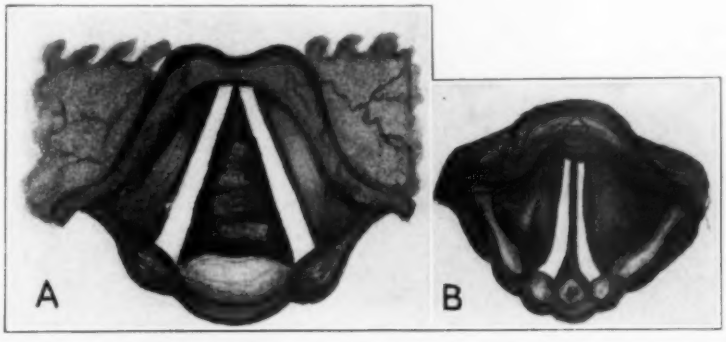

Fig. 1.—*A*, schematic drawing showing the hyperplastic enlargement of the interarytenoid space. *B*, schematic drawing of the larynx during phonation, showing the padlike enlargement of the interarytenoid space interfering with the complete closure of the vocal cords.

DIFFERENTIAL DIAGNOSIS

Laryngeal papilloma is usually multiple, has a cauliflower appearance and is not limited to the interarytenoid space. Pachydermia laryngis is a thickening of the posterior ends of the true vocal cords and histologically consists of thickened epithelium. A malignant tumor usually impairs the mobility of the vocal cords and has a definite histologic nature. Tuberculoma sometimes occurs in the interarytenoid space, but the history, general clinical findings, a Mantoux or Pirquet test and a roentgenogram of the lungs will establish a correct diagnosis. Syphilis can be ruled out by history, other clinical findings and a Wassermann test.

TREATMENT

In the initial stages, when the mucosa of the posterior wall is moderately swollen, electrocauterization, performed with a pointed laryngeal cautery, is

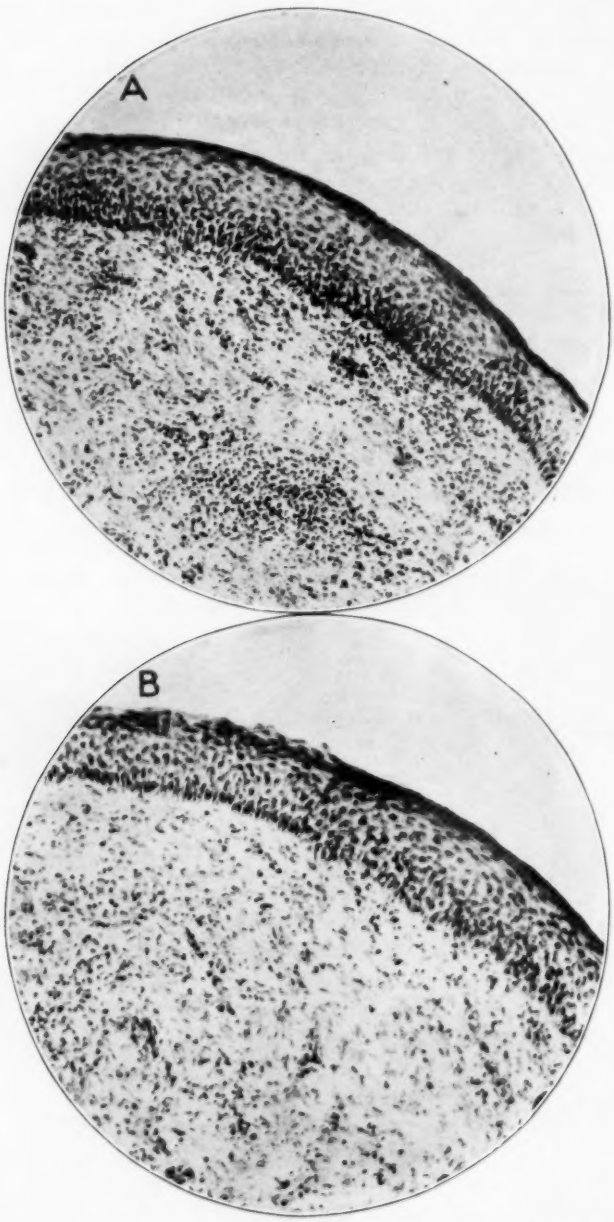

Fig. 2.—*A*, specimen from the interarytenoid space, showing the intact epithelium with the hyperplastic formation of connective tissue. *B*, specimen from the interarytenoid space. At the right side the epithelium is intact. At the left a superficial desquamation begins. Below this there is a hyperplastic formation of connective tissue.

sufficient to produce a shrinkage of the swollen membrane. In cases in which hypertrophy is great and interference with the close contact of the vocal cords is present, excision by the Corde curet is indicated. I favor the indirect method of operation, with local anesthesia induced by a 10 to 15 per cent solution of cocaine. After operation the patient is instructed not to sing for a period of from four to six weeks until the vocal cords become adjusted to the new relationship in the larynx. Complete restoration of the voice occurred in my cases after surgical excision of the hyperplastic enlargement.

REPORT OF CASES

For illustration the following short histories are cited:

CASE 1.—M. M., a lyric soprano, suffered from long-standing catarrh of the throat and larynx. She had a good voice before but could hardly sing at the time of the first examination. Hyperplastic swelling of the interarytenoid space was present, with incomplete closure of the vocal cords, accompanied by hypertrophy of the lingual gland. The general health was normal. The tonsils had

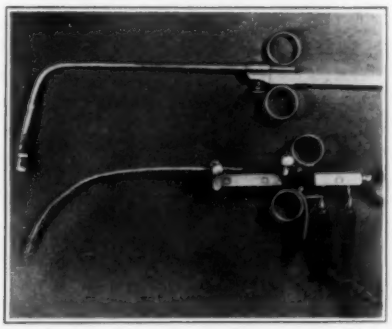

Fig. 3.—Instruments used in electrocauterization and operation.

previously been removed. The hyperplastic infiltration of the interarytenoid space as well as the hypertrophied parts of the lingual gland were removed by the indirect method with the area under local anesthesia. Complete restoration of the voice was obtained, with an increase in diapason and tone.

CASE 2.—I. K., a soprano, came to Los Angeles on a concert tour but was compelled to remain for restoration of her voice because of difficulty in singing. She had been under the care of prominent specialists in the East and in Europe. The pharynx was normal. The mucosa of the larynx and the vocal cords were not inflamed; bluish hyperplastic swelling of the interarytenoid space was present, causing incomplete closure of the vocal cord. The hyperplastic formation was removed by the indirect method with a Corde instrument. A few weeks later the patient sang again in a concert in Los Angeles; the tone of the voice was clear and fresh; the diapason was much greater, and there was no difficulty in singing.

CASE 3.—F. S., a basso, was examined in Los Angeles while on a concert tour. Examination revealed acute congestion in the larynx. The vocal cords were normal in shape, but closure was incomplete because of hyperplastic swelling of the interarytenoid space. Correction was suggested but could not be carried out because of pending engagements in other cities and a trip to Europe.

SUMMARY

1. The hyperplastic laryngitis of singers has definite characteristics and presents a specific clinical entity observed among members of this profession.

2. Restoration of the natural mechanism of voice production can be accomplished by the methods described in the present article.

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COLOR OF THE NASAL SEPTUM

Its Relation to Autonomic Imbalance

EDWIN B. BILCHICK, M.D., NEW YORK

The modern concept of the autonomic nervous system is that of a vast and varied mechanism controlling the vegetative processes of the body. It consists of the ganglions that are anatomically and functionally connected with the central nervous system through the visceral efferent components of the cerebral and spinal nerves and the nerves that arise in these ganglions.[1] It is composed of the sympathetic system, which includes the cervicothoracic and lumbar divisions, and the parasympathetic system, which includes the cranial and sacral divisions. Extensive studies of the autonomic system have been carried out to show its regulation of the so-called vegetative processes in the body: digestion, respiration, circulation, excretion, urination, defecation and secretion. The relation of the autonomic system to changes in the blood pressure, basal metabolism and vascular tonus and to the action of the endocrine glands, particularly the thyroid, pituitary and adrenal glands, forms an interesting chapter in experimental physiology and pharmacology. In 1909 Eppinger and Hess [2] evolved the theory of antagonism of the sympathetic versus the parasympathetic nervous system. In health the resultant status is said to be one of autonomic balance. Predominance of one system is called autonomic imbalance. If the sympathetic system is predominant, there is a state of sympathicotonia; if the parasympathetic system predominates, there is a state of parasympathicotonia or vagotonia. On the basis of this classification patients were put into groups, and therapy was based on this theory. It should be noted that the concept of synergism of the two systems is preferable to that of antagonism. Stimulation with drugs frequently gives reactions opposite to those expected, depending on the dose. Some persons, particularly those with hyperthyroidism, may show hyperirritability of both systems at the same time.

Many authors have attempted to show a definite correlation between autonomic imbalance and the blood chemistry, particularly the acidity or alkalinity of the blood. Others have attempted to show a relation between the autonomic nervous system and the nasal membranes. Jarvis [3] in a series of publications in 1933 and 1934 described his study of the relation between the color of the nasal membranes,

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From the Department of Otolaryngology of the Columbia University and the Presbyterian Hospital Medical Center, Dr. John D. Kernan, Director.

1. Kuntz, Albert: The Autonomic Nervous System: Essential Anatomy, J. A. M. A. **106**:345 (Feb. 1) 1936.

2. Eppinger and Hess: Zur Pathologie des vegetativen Nervensystems, Ztschr. f. klin. Med. **67**:345, 1909.

3. Jarvis, D. C.: Ann. Otol., Rhin. & Laryng. **41**:1124 (Dec.) 1932; Tr. Am. Laryng. A. **54**:112, 1932; Changes in the Color of the Intranasal Mucous Membrane as a Guide to the Status of the Sympathetic and of the Parasympathetic System, Arch. Otolaryng. **21**:131 (Feb.) 1935.

especially of the septum, and the autonomic system. He evolved the concept of the color of the septum as a key to the status of the autonomic system and described several types of syndromes, as follows:

1. Sympathetic dominance, the syndrome of the red septum, resulting from excessive acidity of the blood and from excessive intake of acid-forming foods, such as meat, eggs, fish, sweets and salts. Persons with this syndrome are restless and tired and suffer from frequent staphylococcic and streptococcic infections. They frequently have hyperthyroidism, diabetes and hypertension.

2. Parasympathetic dominance, the syndrome of the pale septum, resulting from excessive alkalinity of the blood and from excessive intake of alkaline ash foods. Persons with this syndrome are subject to angioneurotic edema, itching skin, migraine, asthma, eczema and digestive disturbances.

3. Mixed types. Jarvis described his treatment of these syndromes by prescribing nitrohydrochloric acid and foods forming an acid ash for those with a pale septum and alkaline ash diets for those with a red septum. He reported excellent clinical results. Blackmar,[4] working from Jarvis' conclusions, evolved the concept of "shifting the autonomic level"; that is, changing the status of autonomic imbalance by using the color of the septum as the guide to treatment. He regarded allergic conditions as due to a state of alkalosis. He expressed the belief that staphylococcic and streptococcic infections are due to a state of sympathicotonia. Frank,[5] accepting Jarvis' theories, treated patients with nitrohydrochloric acid with good results. Beckman reported the obtaining of relief from symptoms of hay fever by treatment with acid in 40 per cent of his cases. Alden[6] recommended treatment of allergic conditions with acids.

In order to determine the value of this new concept Bernheimer,[7] of Chicago, studied fifty patients with disturbance of the acid-base equilibrium. These included persons with such conditions as nephritis, diabetes and toxemia. He found no relation between changes in the color of the septum and the acid-base equilibrium as evaluated by the p_H of the blood and urine. He also studied one hundred normal persons but was unable to classify them by type as judged by the color of the septum. No abnormal variations in the p_H of the blood were found in patients with vasomotor rhinitis, in which one would expect alkalosis. Furthermore, treatment of these patients with acids and with acid ash diets gave no results as judged by clinical status and the color of the septum. Jarvis[8] then decided that this theory is untenable but concluded that his therapeutic results were due to the action of minerals, drugs, hormones and vitamins on the vasomotor mechanism of the nose.

Bernheimer[9] in another series of studies described the effect of various drugs, salts, vitamins and gland extracts on the color of the nasal septum. He found no demonstrable permanent effect, all results being transitory. Moreover, in a group of cases of systemic vascular disease he found no corresponding change in the color of the septum. Because of the interest aroused by these reports and the conflicting findings, it was decided to institute a preliminary series of obser-

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4. Blackmar, F.: Laryngoscope **44**:959 (Dec.) 1934.
5. Frank, I.: Laryngoscope **43**:48 (Jan.) 1933.
6. Alden, A. M.: Tr. Am. Laryng. A. **54**:118, 1932.
7. Bernheimer, L. B., and Cohn, D. J.: Color of the Nasal Septum: Critical Study of Its Supposed Value as an Index for Dietary Therapy in Diseases of the Upper Respiratory Tract, J. A. M. A. **100**:1324 (April 29) 1933.
8. Jarvis, D. C., quoted by Bernheimer.[9]
9. Bernheimer, L. B.: Color Index of the Nasal Septum: Critical Study of the Vasomotor Mechanism of the Nose, Arch. Otolaryng. **20**:31 (July) 1934.

vations to determine whether any relation could be discovered between the color of the septum and the blood pressure, red cell count, blood chemistry, basal metabolism, systemic disease or constitutional type as determined by Draper.[10] One hundred ward patients of the Presbyterian Hospital were included in this study. To avoid the confusing effects of season one group of patients was examined in summer and another in winter. Those with infection or a pathologic condition of the upper respiratory tract were eliminated, as were also those with severe anemia or allergic conditions. In this series there were included patients with diabetes, nephritis, hypertension, cardiac disease, hyperthyroidism, hypothyroidism and rheumatism.

Comparison of the records of these patients with the results of study of their septal membranes and in some cases with their constitutional type enabled me to determine that in this series of cases the following conclusions could be drawn: 1. There is no demonstrable relation between the color of the nasal septum and the blood pressure. 2. There is no relation between the color of the septum and the nitrogen or cholesterol content of the blood. 3. In diabetic patients, no relation can be found between the color of the septum and the blood sugar. 4. Except in severe anemia, the color of the septum bears no relation to the red blood cell count. 5. The color of the septum bears no relation to the basal metabolic rate.

Autonomic imbalance is frequently expressed in gastro-intestinal dysfunction and disease. It has been shown that the "ulcer type" is definitely vagotonic while the "gallbladder type" is sympathicotonic. Draper described these two types with their anatomic and physiologic characteristics in his studies of the constitutional types.[10] Jarvis' work presupposed that persons with sympathicotonia show an increased acidity of the blood, and he based his therapeutic dietetics on this assumption, prescribing an alkaline ash diet for them. This is in accordance with a great deal of popular clamor today for alkalinization as a remedy for many ailments, particularly arthritis. Several German investigators, among them Balint[11] and Cespai, Hollo and Weiss,[12] on the other hand, have shown that sympathicotonia is associated with alkalinity of the blood. Experimental work on animals and man showed shifting toward alkalinity to be accompanied by an increase in tonus of the sympathetic nervous system. Balint stated that patients with gastric ulcer, who are typically vagotonic, showed hyperacidity of the blood. Patients with hyperthyroidism were shown to have alkalosis. When their condition improved, there was a shift toward acidity. Thus, the concept of hyperacidity as the cause or concomitant of sympathicotonia is untenable. The syndrome of the red nasal septum associated with sympathicotonia and hyperacidity cannot be substantiated.

SUMMARY

The supposed relation between autonomic imbalance and the color of the septum is analyzed.

Bernheimer's observations showing the absence of relation between the color of the septum and autonomic imbalance are confirmed.

In a series of one hundred cases no relation was found between the color of the septum and blood pressure, blood sugar, blood nitrogen, basal metabolic rate or red blood count (except in severe anemia).

876 Park Avenue.

10. Draper, George: Human Constitution, Philadelphia, W. B. Saunders Company, 1924.

11. Balint, R., quoted by Kuntz, A.: The Autonomic Nervous System, ed. 2, Philadelphia, Lea & Febiger, 1934, p. 482.

12. Cespai, Hollo and Weiss: Ztschr. f. d. ges. exper. Med. **56**:206, 1925.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

TONSILS AND ADENOIDS

FRENCH K. HANSEL, M.D.
ST. LOUIS

(Concluded from page 661)

TREATMENT

Discussing nupercaine as a local anesthetic in otolaryngology, Egan [82] states that he has found that a 1 : 1,000 concentration appears to be a satisfactory anesthetic for infiltration previous to tonsillectomy. The duration of the anesthesia produced exceeds that produced by 1 per cent procaine hydrochloride. No evidence of local irritation, delayed healing or systemic intoxication was noted after the use of nupercaine ciba.

A new soft palate retractor and an adenoid punch for removing the remnants of adenoids were devised by Love.[83] By means of the soft palate retractors a direct view of the epipharynx can be obtained. When remnants of adenoids are present, they can be removed easily with the basket punch. The soft palate retractor can also be used when inserting a postnasal pack.

A new soft palate retractor permitting adenoidectomy under direct vision was devised by Gidoll.[84] The instrument consists of a modification of the Greene mouth gag. Utilizing the old principle of a nasal catheter in each nostril, it has the new feature of an attachment to the mouth gag which anchors the catheters in place by means of thumbscrews. The attachment is constructed at an angle of 45 degrees from the vertical, so as to permit easy access to the nostrils or the throat.

A ligature carrier and tier were designed by Harris.[85] The instrument consists of a straight clamp with a sliding hook on one shank to which one end of the ligature is attached. The bleeding point is first fixed with a regular tonsillar artery clamp. A slip-knot is made, and

82. Egan, E. F.: Nupercaine as a Local Anesthetic in Rhino-Laryngology, Laryngoscope **44**:829, 1934.

83. Love, A. A.: A New Soft Palate Retractor and Adenoid Punch, Arch. Otolaryng. **19**:66 (Jan.) 1934.

84. Gidoll, S. H.: A New Soft Palate Retractor Permitting Adenoidectomy Under Direct Vision, Arch. Otolaryng. **20**:69 (July) 1934.

85. Harris, M. L.: Ligature Carrier and Tier, Arch. Otolaryng. **19**:68 (Jan.) 1934.

the fixed end is caught by the instrument close to the knot. The long end is slung about the sliding hook and tied. The loop is then slipped over the clamp with one hand manipulating this clamp on the bleeding point while the other holds the instrument to the clamped tissue. The loop is pulled tight with the sliding hook, as in manipulating the ordinary snare.

A new instrument for the treatment of peritonsillar abscess was designed by Goldman.[86] It consists of a sharp-pointed lancet cannula, which can be attached to a suction apparatus. After the contents of the abscess cavity have been evacuated, the cannula is slowly withdrawn. As the sharp end reaches the surface, it is used as a lancet to convert the puncture into an incision providing more adequate and sustained drainage.

Macpherson [87] describes an operation for the removal of carcinoma of the tonsil and contiguous parts. He adds that several modifications of the operation may be employed, for example, the use of radium before and after the removal of the diseased tissues. The entire operation may be done at one time, with ligation of the internal carotid artery. The lingual, facial and ascending pharyngeal arteries may be tied, leaving the external carotid artery itself patent.

The progress which has been made in surgical treatment of the tonsils was illustrated by Fowler [88] by slides and moving pictures with a background of 25,000 cases.

Discussing the matter of results of tonsillectomy in private practice, Bass [89] concludes, after a careful analysis of 150 cases, that when the greatest care was exercised in determining indications for operation and the choice of the operator, the children were distinctly benefited in 88 per cent of the cases.

Personal observations on the after-effects of tonsillectomy are reported by Mason [90] also. The report is based on an analysis of the histories of 84 patients observed in private practice. From his observations he concludes that enlarged tonsillar lymph nodes, tonsillitis, especially when repeated, and rheumatic fever are all direct indications for tonsillectomy. Frequent prolonged colds, sinusitis, otitis and nasal

86. Goldman, I. B.: A New Instrument for Treatment of Peritonsillar Abscess, Laryngoscope **45**:964, 1935.

87. Macpherson, D.: Operation for Removal of Carcinoma of the Faucial Tonsil and Contiguous Parts, Ann. Otol., Rhin. & Laryng. **43**:717, 1934.

88. Fowler, R. H.: Progress in Tonsil Surgery, Illustrated by Slides and Motion Pictures, with a Background of Twenty-Five Thousand Cases, Laryngoscope **44**:769, 1934.

89. Bass, M. H.: Results of Tonsillectomy in Private Practice, Laryngoscope **44**:780, 1934.

90. Mason, H. H.: Personal Observations on the After-Effects of Tonsillectomy, Laryngoscope **44**:784, 1934.

obstruction demand an investigation of the adenoids and their removal if they are enlarged or so placed that they cause trouble. He does not believe that every adenoidectomy should be accompanied by tonsillectomy, especially in young children.

Improved technic in tonsillectomy is advocated by Holding.[91] He discusses the indications and contraindications, anesthesia, methods of operation, position of the patient and the use of electrocoagulation. He states that the general surgeons and general practitioners will continue to perform tonsillectomies until the majority of otolaryngologists justify their specialty by performing better operations.

Discussing the tonsil problem, Jones [92] outlines the methods by which failures in the results of tonsillectomy may be obviated.

According to the experience of Hays,[93] the usual types of treatment in the form of sprays or gargles for discomfort and pain in the throat, either primarily or after tonsillectomy, seldom reach the inflamed areas and therefore are of little use. He advocates the use of chewing gum, particularly that medicated with acetylsalicylic acid. The increased flow of saliva resulting from the chewing of gum causes the inflammation to heal more quickly. The small amount of acetylsalicylic acid swallowed brings about the usual beneficial reactions to that drug—deadening of pain.

In a comprehensive article Yonker [94] discusses the obstacles encountered in electrocoagulation of the tonsils. An unbiased investigation was carried out on the use of this method. The results obtained were reported as unsatisfactory, and Yonker and his associates are still in doubt as to whether there are any indications for this method of removal of tonsils. In previous studies, Shambaugh, Dougherty and Yonker [95] reported their experiences with electrocoagulation in the practice of otolaryngology as it was investigated in the department of otolaryngology of the Rush Medical College. Since that report they have continued to follow up those patients, and they find no reason for altering their opinions in respect to the conclusions there expressed.

Among the difficulties encountered by this method, the following were enumerated: pain occurring during and after treatments; the length of time the patient must be under treatment, causing an unneces-

91. Holding, A. F.: The Need for Improved Technic in Tonsillectomy, Laryngoscope **45**:458, 1935.

92. Jones, D. H.: How to Obviate Failures in the Results of Tonsillectomy, Laryngoscope **44**:847, 1934.

93. Hays, H.: Control of Discomfort and Pain in Throat Affections and After Tonsillectomy, Eye, Ear, Nose & Throat Monthly **15**:194 (July) 1936.

94. Yonker, W. J.: Obstacles Encountered in Electrocoagulation of Tonsils, Ann. Otol., Rhin. & Laryng. **43**:1117, 1934.

95. Shambaugh, G. E.; Dougherty, C. L., and Yonker, W. J.: Electrocoagulation in the Practice of Otolaryngology, S. Clin. North America **17**:1157, 1932.

sary delay in the removal of pathologic tonsils; the complication of secondary bleeding, and especially the uncertainty as to whether all pathologic tissue has been removed. In the series of cases reported by Yonker, the investigators were not satisfied that all tonsillar tissue had been removed from a single patient. Patients were examined who had had electrocoagulation done elsewhere, and in not a single instance was it found that the tonsils had been eradicated. It was finally concluded that because of the various obstacles enumerated, this method of removing tonsils has thus far been disappointing.

Roberts [96] sketches briefly the rise of diathermy to favor in the treatment of tonsillar sepsis and its present position in specialist opinion in the United States.

The good results obtained by diathermy in malignant disease of the tonsil suggest that it might be used for the septic tonsil. The idea was elaborated by the instrument makers, who developed a suitable apparatus. "Ushered in by clever advertising and marketed by good salesmanship and later by the Life Extension Institute, who are advocating its use, there arose a public demand for this form of treatment utterly out of proportion to its merits." Claims are made that the method is safe and simple; that there is less pain and less risk of hemorrhage; finally, that it saves the patient time and money.

The method has been widely used in the United States, and Roberts has had a mass of material on which to base his opinions. They are uniformly unfavorable. He points out first the difficulty of removing all the tonsil. Even with bipolar needles the area coagulated is pyramidal, with the base to the faucial surface; the difficulty of complete removal is compared to taking custard out of a cup with a spoon; ridges and streaks are left between the spoonfuls. In practice, the danger of secondary hemorrhage has proved to be considerable, and when hemorrhage does occur it is difficult to control. In many cases there is considerable postoperative pain. Lastly, in many cases the removal is incomplete. In support of these findings Roberts quotes the stated opinions of various clinics, all unfavorable, and many (for example, those of the Shambaugh school) violently so.

> Contrary to the advice freely given by some, that surgical diathermy is the procedure of choice over tonsillectomy, the facts are that with the use of surgical diathermy:
>
> Theoretically, the tonsil is not entirely removed and if my idea of the mechanics of this technique is correct, it cannot be. Practically, as most of us see end results, this is the fact.
>
> The danger of secondary hemorrhage is great.
>
> There is postoperative pain in the series of treatments.

96. Roberts, E. H.: The Present Status of Diathermy or Electrocoagulation in the Treatment of Tonsil Disease, Laryngoscope **44**:941, 1934.

Secondary infection, with edema, is always present in some degree, and scar tissue invariably is the end result.

Diathermy is unsurgical and dangerous in itself, and may be dangerously inefficient from the standpoint of the patient.

Additional surgical removal is necessary afterwards.

Tonsillectomy is, as yet, the only operation which adequately fulfills the demand for complete removal of the faucial tonsil and complete eradication of this organ as a focus of infection.

According to Mock,[97] tonsillectomy by electrocoagulation has no doubt been abused.

Dillinger, the champion of electrocoagulation for the removal of the tonsils, says: "This method requires more skill and training than the ordinary surgical tonsillectomy, and no one except a throat specialist should attempt to use diathermy in the throat."

Beals speaks of the general practitioner being misled, because he believes he can easily master this apparently simple but in reality arduous method and decries the unethical manufacturer for adding chaos by sending out high pressure salesmen to sell to any one for the sake of a sale.

Coates, in discussing this paper, advocated that the laryngologist should include electrocoagulation to keep it out of the hands of those who are not qualified. Myers reported thirty casualties and says that it is no method for the neophyte.

When the public learns of the dangers and complications that may occur during the removal of tonsils by electrocoagulation, and, what may be even worse, leaving a buried focus of infection from an incomplete removal, it will shun this operation as it would a rabid dog, and the laryngologist will have difficulty in persuading the person with an otherwise inoperable condition to accept this method as the operation of necessity.

Occasionally cases are referred in which there is a history of advanced tuberculosis, exophthalmic goiter or myocardial degeneration, requiring the removal of the tonsils. Electrocoagulation is the salvation of these patients.

According to Norrie,[98] removal of tonsils by diathermy is far from being the ideal method in the average case of tonsillar infection and with the present technic should be reserved for cases in which dissection is contraindicated. He further states that the treatment of tonsils by diathermy involves problems which never arise in operation and which on occasion may tax the ingenuity of the operator.

Jobson [99] has designated a set of instruments for electrosurgical tonsillectomy which is employed in adults, preferably under local anesthesia. The set of instruments consists of an insulated snare furnished with a no. 7 wire, a pair of tonsil forceps, a spud-shaped dissector for freeing

97. Mock, H. E.: Electrosurgery: A Discussion of Indications, Advantages, Disadvantages and Warnings Concerning Its Use, J. A. M. A. **104**:2341 (June 29) 1935.

98. Norrie, F. H.: Notes on Diathermy in Ear, Nose and Throat, J. Laryng. & Otol. **49**:73, 1934.

99. Jobson, G. B.: A Set of Instruments for Electrosurgical Tonsillectomy, Arch. Otolaryng. **20**:707 (Nov.) 1934.

the mucous membrane from the tonsil and an insulated tongue depressor. An endothermy machine furnishes the cutting current. With the tonsillar area under local anesthesia, the patient sits on a wooden chair with the inactive electrode in contact with the bare buttocks. If the patient is lying on a metal table, the top should be insulated with rubber mats. The tonsil is seized with the forceps and drawn toward the median line. After the mucous membranes are dissected free around the tonsil, the loop of the snare wire, which is the active electrode, is applied over the forceps, which remains attached to the tonsil, and then is carried to and placed beneath the inferior pole of the tonsil with the tip of the snare in the supratonsillar fossa. The tonsil is drawn toward the midline; the snare is compressed, and the current is turned on with a foot switch. The tonsil comes away, leaving a dry fossa. With this procedure, practically a bloodless operation, with slight edema, less postoperative pain and less scarring may be obtained than by any other method. Of 358 cases, Jobson encountered slight secondary hemorrhage in 3.

Malone [100] describes an electrosurgical technic of tonsillectomy under local anesthesia, using the high frequency undamped electric cutting current. Any bleeding occurring during the dissection can be checked promptly by switching the machine to a damped, or coagulating, current and touching the bleeding point.

Since 1929 Hasenjäger [101] has treated 47 patients with subacute and chronic tonsillitis by means of roentgen rays. In those cases in which there is a moderate amount of inflammation, he uses a dose of from 20 to 25 per cent of a unit skin dose, and in more chronic cases, as much as 40 per cent. Most of his patients were treated only once, although several were treated two or three times at intervals of from four to six weeks. Of 32 cases which he was able to follow up, he believes that there was a cure in 23, improvement in 5, and no improvement in 4. Half the patients were children between the ages of 4 and 15 years. Roentgen therapy can be used for all patients for whom surgical intervention is contraindicated, save perhaps for children under 2 years.

Hasenjäger treated 15 patients with acute tonsillitis, lymphadenitis or peritonsillitis and seemed to get satisfactory results. The danger of increased swelling and possible edema of the glottis seems slight even in the treatment of patients with severe conditions, unless edema of the glottis is already present.

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100. Malone, P. W.: Electrosurgical Technic of Tonsillectomy Under Local Anesthesia, Arch. Otolaryng. **21**:93 (Jan.) 1935.

101. Hasenjäger, E.: Roentgenotherapy of Tonsillitis, Med. Klin. **29**:1311, 1933.

In cases of chronic tonsillitis with recurrent acute angina, Hess [102] successfully employed roentgen therapy in giving permanent as well as immediate relief from symptoms. Among 31 of 40 cases, the following results were obtained: permanent cure, 68 per cent; cure with recurrence, 10 per cent; improved, 12 per cent, and unchanged, 10 per cent. The beneficial effect of the roentgen rays is produced by the destruction of lymphatic cells. It is believed that the disintegrated cells stimulate the surrounding tissue to increased bactericidal activity and in this way the infection is overcome. One-third erythema dose for the superficial tissues and one-fifth erythema dose for the deep tissues were used. The rays were directed from behind upward and mesially under the angle of the mandible.

Wolff [103] reports 22 cases, mainly in young men, in which tonsillectomy was done after roentgen treatment of one tonsil with protection of the other. Anatomicopathologic examination revealed no noteworthy difference between the irradiated and the nonirradiated tonsil.

Niemeyer [104] administered roentgen treatment to 54 children with severe recurrent infections of the tonsils and the lymphatic tissue of the nasopharynx. Two thirds of them were free from recurrence for two and one-half years, and in the remaining third the infections which developed were much milder than those which occurred previously. The general health of the children was also improved.

Cutler [105] limits his discussion to observations on the effect of the total period of irradiation on the skin, the bed of the tumor and the local lesion in carcinoma of the tonsil, pharynx and larynx treated by the 4 Gm. radium pack. He selected 5 cases, which are of interest as evidence in the problem of the upper limit for total irradiation without the production of immunity to radiation and as evidence of the dose necessary to produce epidermitis and of the upper limits of safety in massive radium therapy. The lesion was early and localized in 1 case and advanced in 4. Disappearance of the lesions was produced in all the cases by protracted radium pack therapy. The lesions were exposed twice daily and consecutively. The distances were 10 and 15 cm. Epithelitis was produced in every instance and epidermitis in 3 of the 5 cases. These observations are presented to demonstrate a primary

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102. Hess, P.: Roentgenotherapy in Chronic Tonsillitis, München. med. Wchnschr. **80**:384, 1933.

103. Wolff, E.: Roentgen Treatment in Chronic Tonsillitis with Especial Regard to Anatomicopathologic Changes in Irradiated Tonsils, Finska läk.-sällsk. handl. **75**:965, 1933.

104. Niemeyer, R.: Roentgen Treatment of Recurrent Inflammation of the Nasopharynx, Monatschr. f. Kinderh. **58**:439, 1933.

105. Cutler, M.: Radium Dosage and Technic in Carcinoma of Tonsil, Pharynx, and Larynx, Am. J. Roentgenol. **33**:690, 1935.

disappearance of disease in response to protracted irradiation. The patients have been free from disease for periods varying between nine and eighteen months. The short interval since treatment permits no deductions on the permanence of the results. These observations, however, seem to confirm the possibility that if continuity of treatment is observed and the total dose is adequate the total period of irradiation may be prolonged to forty, fifty or even sixty days without necessarily inducing immunity to radium. Although it is possible that local recurrences may still develop in these cases because of the short interval, it is much more likely that reappearance of the disease—if it occurs—will take place in the form of metastases to lymph nodes outside the field of irradiation or to distant viscera.

Comprehensive reports on irradiation therapy and malignant tumor of the tonsils are reviewed in the reports of Martin [106] and Galloway.[107] The technic and the results obtained are thoroughly discussed.

COMPLICATIONS

As a result of his studies of bacteremia and acute infection of the upper respiratory tract, Ballenger [108] concludes that a transitory or an evanescent type of bacteremia is fairly common and possibly is the usual complication following acute infection of the upper respiratory tract, especially that due to hemolytic streptococci. He further states that unexplained fever or fever out of proportion to the degree of the involvement of the throat can be explained by this probable bacteremia. If bacteremia is present, possible complications are limited only by the distribution of the blood stream.

Baum [109] suggests a method of specific treatment in certain streptococcic infections. His preliminary studies suggest that blood serum from patients convalescing from various streptococcic infections frequently contains immune substances antagonistic to similar but not necessarily identical organisms. Specificity seems to be indicated by positive agglutination reactions. Moderate doses of such serums, properly checked for specificity, or blood transfusions from their donors have proved highly effective in the treatment of many varieties of streptococcic infection. Further tests of the method are in progress

106. Martin, C. L.: Irradiation Therapy of Malignant Tumors of the Oral Cavity, Eye, Ear, Nose and Throat, Ann. Otol., Rhin. & Laryng. **44**:426, 1935.

107. Galloway, T. C.: The Selective Treatment of Malignancy About the Head, Ann. Otol., Rhin. & Laryng. **44**:450, 1935.

108. Ballenger, H. C.: Bacteremia and Acute Infections of the Upper Respiratory Tract, Arch. Otolaryng. **20**:452 (Oct.) 1934.

109. Baum, H. L.: A Method of Specific Treatment in Certain Streptococcic Infections, Arch. Otolaryng. **20**:504 (Oct.) 1934.

in addition to a study of similar properties of serums from other non-streptococcic infections.

Havens [110] made a study of 125 unselected cases of suppurative processes in the neck. There were 103 cases of acute suppuration, 18 of broken-down tuberculous lymph glands and 2 of infected branchial cysts and also 2 of Bezold abscesses. It is noteworthy that in the group of 103 cases of acute suppurative conditions, sore throat and tonsillitis were responsible in 8, tonsillectomy in 8 and retropharyngeal abscess in 2.

In the opinion of Truffert and Viela,[111] all suppurations near the posterior pillar of the fauces or the floor of the mouth tend to point toward the hyothyro-epiglottic space, between the thyrohyoid membrane and the anterior face of the epiglottis. Acute abscesses in this region may require opening below the hyoid, unless pointing upward occurs early. Diffuse forms may become extremely grave, with extreme dysphagia and a croaking voice.

Laryngeal abscess is primarily influenzal or traumatic; secondarily, from extension of neighboring suppuration or secondary infection of a specific perichondritis. It is most frequent in the epiglottis, next in the arytenoids and secondarily in the posterior wall of the larynx or the pharyngolaryngeal gutter. Tuberculosis, syphilis, cancer or necrosis from physical agents used in treatment (radium) are blamed for perichondritis with abscess.

Retrolaryngeal and pharyngeal abscesses, when acute, are due to glandular infection or to trauma of the pharyngeal musculature and mucosa. If a good-sized collection of pus can be localized internally, it should be widely opened within, to avoid the danger of severe delayed hemorrhage. If this is not possible and roentgenograms show definite localization, lateral cervical drainage must be made. Cold abscesses of this region are always tuberculous, arising from the neighboring vertebrae, and require lateral opening, with orthopedic immobilization of the cervical portion of the spine.

The diagnosis and treatment of parapharyngeal infection and of thrombosis of the internal jugular vein were reported by Beck.[112] According to his observations, the exact diagnosis of deep infections in the neck necessitates a consideration of the site of the initial infection or portal of entry, a thorough understanding of applied anatomy

110. Havens, F. Z.: Infections of the Neck, Arch. Otolaryng. **21**:536 (May) 1935.

111. Truffert, P., and Viela, A.: Perilaryngeal and Pharyngeal Suppurations, Oto-rhino-laryng. internat. **18**:677, 1934.

112. Beck, A. L.: Parapharyngeal Infections and Internal Jugular Vein Thrombosis: Diagnosis and Treatment, Laryngoscope **44**:431, 1934.

and a familiarity with regional manifestations, which can more or less be grouped anatomically. The common cause or starting point of infections in the neck is inflammation in or about the tonsils. Other conditions which may result in infections in the neck are described. Beck points out that the occurrence of a distinct complicating infection may be regarded as a positive indication for immediate surgical drainage of the neck and also for resection of the internal jugular vein.

Waldapfel [113] differentiates two types of tonsillogenic sepsis. In the first type, dissemination takes place directly by way of the blood stream from small thrombosed tonsillar veins and advances in the direction of the jugular vein. In the second type dissemination takes place by way of the lymph channels along the neck, and from there by way of periphlebitis, usually originating in a lymph node, with subsequent invasion of the blood stream and with secondary thrombosis of the jugular vein. Another important factor is that the port of entry is not always identical with the septic focus. The infection may originate in the tonsil, but the septic focus is frequently in the loose tissue of the parapharyngeal space. The therapy should take account of these pathologic anatomic conditions. The author has made it a rule to open and drain the parapharyngeal space from the outside and to examine the jugular vein whenever there is the slightest suspicion of infection. If the local changes indicate that the septic focus is within the tonsil, it is removed. By this method he has succeeded in greatly reducing the mortality rate. The clinical symptoms that indicate the infection of the parapharyngeal space are infiltration of the neck, sensitivity to pressure in the region of the jugular vein, attacks of chills and symptoms of general sepsis. If these symptoms are apparent, the diagnosis and the decision to resort to operation ought to present no difficulties. On the other hand, when the local changes seem insignificant, it may be difficult to diagnose the condition. Waldapfel calls attention to the jugular glands as septic foci and stresses that they ought to be removed even if there is only an inflammatory swelling.

Uffenorde [114] reports and discusses several cases. The first illustrates the process of transmission of the infection from the tonsils by way of the lymph vessels to the regional glands in a severe form as the possible beginning of thrombosis of the jugular and facial veins and sepsis. In this case the tonsillitis with glandular swelling, which had been neglected by the patient, had produced a considerable number of adhesions between the inflamed glands and the vascular sheath and

113. Waldapfel, R.: Clinical Aspects and Therapy of Sepsis Following Tonsillitis, Wien. klin. Wchnschr. **48**:136, 1935.

114. Uffenorde, W.: Thrombosis of the Jugular Veins as a Complication of Tonsillar Infection Transmitted by Way of the Lymph Vessels, Ztschr. f. Laryng., Rhin., Otol. **25**:307, 1934.

the jugular and facial veins, but thrombophlebitis and sepsis had not developed. Above the adhesions an encapsulated abscess had developed in the parapharyngeal space. This abscess was separated from the peritonsillar inflammation. This is possible because the lymph vessels pass from the tonsil through the peritonsillar tissue and the lateral pharyngeal wall into the parapharyngeal space and farther to the main regional gland at the site of convergence of the vena jugularis and the vena facialis communis. In such cases, with an encapsulated abscess of the parapharyngeal space, there is great danger of erosion of the vessels, but in this instance perivascular indurations had protected the vessels and thus had prevented sepsis. Uffenorde thinks that his method of opening the parapharyngeal space is advisable. He emphasizes that the space should be entered, not from above, but from below the digastric muscle and along the internal jugular vein. Along the jugular vein the infection spreads from the parapharyngeal space to the mediastinum, and along this same route the lymph nodes are located. This path is the more suitable, since the thrombosis of the jugular vein, a rather frequent complication, can be watched from it.

The second case illustrates the further course of the transmission of the infection by the lymph channels with secondary invasion of the blood stream and subsequent sepsis. Uffernorde reports three other cases and in the conclusion emphasizes that posttonsillitic sepsis develops by way of the lymph channels either directly or, more often, indirectly by spreading of the regional lymphadenitis with subsequent thrombosis of the jugular and facial veins. The treatment consists in complete enucleation of the palatine tonsils, opening of the vascular sheath, with removal of the inflamed glands, careful inspection of the veins and opening of the parapharyngeal space. If the veins are thrombosed, they are completely excluded.

Haymann [115] found that in 12,500 necropsy records examined, death following tonsillar sepsis was reported in about 5.3 per thousand. In general, in the presence of bilateral but unequal tonsillar infection, sepsis is more likely to develop in the more seriously involved side. In about 30 per cent of the cases no recognizable harmful condition of the tonsils appeared to exist, but general sepsis resulted. In about 35 per cent peritonsillar or retropharyngeal abscesses were present.

Haymann suggests a method of classifying the mode of origin of tonsillogenous sepsis: (1) by direct entrance of the causative agent into the blood stream without preexisting thromboplebitis from isolated thromboses of the retrotonsillar veins or from a progressive jugular

115. Haymann, L.: Surveys on Tonsillogenous Sepsis (Contribution to Pathogenesis, Pathology and Clinical Aspects), Ztschr. f. Hals-, Nasen- u. Ohrenh. **35**: 288, 1934.

thrombosis; (2) by indirect entrance into the blood stream by way of lymphangitic or lymphadenitic changes and (3) by phlegmonous changes, especially in the interstices of the neck, with secondary thrombosis of the veins, especially the jugular.

Myers [116] reports 3 cases of pyemia following acute infection of the tonsils. The first case was that of a white man aged 22, whose illness began with acute tonsillitis followed by peritonsillar abscess. The subsequent course was that of generalized sepsis. The site of thrombophlebitis was not determined. There were septic metastases to the lungs, pleurae, pericardium, wall of the chest, right buttock and right ankle. An anaerobic streptococcus was grown from the pus several times from different locations. The patient died forty days after the onset of the sore throat. In the second case thrombosis of the left internal jugular vein developed subsequent to acute tonsillitis in a youth aged 16. There were metastatic septic lesions in the lungs and pleurae and signs of venous stasis in the basal cerebral ganglions. Streptococcus haemolyticus was recovered from the pleural fluid. The course led to death three and one-half weeks after the onset of the sore throat. The third case was that of a man aged 36, who acquired a left peritonsillar abscess in the wake of an attack of acute tonsillitis. The course of the illness was one of sepsis with an abscess of the right buttock and urinary signs of involvement of the kidney. Streptococcus viridans was recovered from the blood. The patient died six weeks after the onset of the sore throat.

Abt [117] presents an interesting discussion of postanginal sepsis and also reports 4 cases in children. He states that the prognosis of postanginal sepsis is grave. Uffenorde collected reports of 137 cases; only 40 of the patients recovered. These were cured by operation. In Waldapfel's experience, 25 of 43 cases ended fatally. Seventeen of the patients with fatal sepsis were operated on, and 8 were not. Klaus reported 14 deaths in 28 cases. According to Abt, the earlier the purulent focus is detected and the contents evacuated, the more favorable the prognosis. When symtoms of general sepsis are present, ligation of the jugular vein on the infected side as soon as possible is advisable, in order to prevent the formation of metastatic foci throughout the various tissues and organs of the body.

Schneider [118] points out the similarity between posttonsillitic septicemia and puerperal sepsis in respect to etiology, pathogenesis, clinical

116. Myers, W. K.: Posttonsillitic Pyemia: Report of Three Cases, M. Ann. District of Columbia **3**:279, 1934.

117. Abt, I. A.: Postanginal Sepsis, Ann. Otol., Rhin. & Laryng. **43**:441, 1934.

118. Schneider, F. A.: Identity of Course of Posttonsillitic Sepsis and Puerperal Sepsis, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **137**:75, 1933.

course, complications and therapy. In both diseases the infection may spread by way of the following routes; the hematogenous, the lymphogenous, the lymphohematogenous and the perivascular, phlegmonous route. In both forms of sepsis there is the danger of pyemia; metastases to the joints, kidneys, lungs and endocardium may also occur. In both diseases the phlegmonous process tends to invade the large cavities of the body, causing peritonitis in puerperal infection and mediastinitis in posttonsillitic sepsis. Therapy in both forms of sepsis aims at elimination of the primary focus of infection, exposure of infected interstices and incision of peripheral abscesses. Ligation of veins in both diseases is indicated only in the event of thrombosis in those cases in which the infection tends chiefly toward endophlebitic extension. The percentage of cures in cases of severe involvement in which surgical intervention is employed is about 50 per cent for both forms of sepsis.

Hybasek [119] discusses the various types of tonsillitis from the clinical, etiologic, symptomatic and diagnostic aspects and cites the contemporary literature. He classifies tonsillitis as follicular, phlegmonous, pseudomembranous and vesicular. The therapeutic measures used are outlined. A series of reports of cases is presented, and the prognosis is discussed. The résumé of active treatment is as follows: Drainage in acute sepsis is more important than tonsillectomy. The indications for tonsillectomy should be limited rather than amplified in postanginal sepsis. Peritonsillar abscesses should be opened widely orally, and tonsillectomy should not be performed in the presence of a peritonsillar phlegmon. Prophylactic ligation of the jugular vein should not be done, but thrombosed vessels should be ligated or excised. The deep structures of the neck and the parapharyngeal regions should be opened widely and drained. A bibliography is appended.

Interest has been renewed in the danger of tonsillectomy for peritonsillar abscess in the acute inflammatory stage. Several authors consider the so-called abscess tonsillectomy as without danger and strongly recommend it. Four cases have been reported in which serious complications followed tonsillectomy for peritonsillar abscess in the acute stage. Zöllner [120] observed 90 patients on whom tonsillectomy for peritonsillar abscess had been performed. One had a chill and a rise in temperature to 40.5 C. (104.9 F.) eight hours after the operation. Another had a phlegmonous infiltration of the base of the tongue. In comparing the patients on whom tonsillectomy had been performed with those who had had simple incision of the abscess, Zöllner noted an

119. Hybasek, J.: Sepsis Following Angina and Tonsillectomy, Časop. lék. česk. **71**:993, 1932.

120. Zöllner, F.: Tonsillectomy in the Presence of Acute Infection, München. med. Wchnschr. **81**:634, 1934.

immediate postoperative drop in temperature more often in those who had had the simple incision, and febrile episodes more often in those subjected to tonsillectomy. Zöllner recommends simple incision in early peritonsillar abscess and in cases in which the abscess is well walled off. He advises tonsillectomy when there are signs of sepsis, when pus is not obtained after incision and when fever persists after simple incision.

Hofer and Motloch [121] also review the danger of performing tonsillectomy when peritonsillar abscess is present. The importance of this question is due to several factors. Simple incision in cases in which the abscess lies posteriorly and deep is often insufficient, the pus draining poorly through the incision. Hemorrhage into the abscess cavity from the incision wound occurs not infrequently. The tonsils sometimes act like a foreign body in preventing complete drainage of the abscess. In diffuse phlegmonous processes wide open incision and drainage only will suffice. In more than 30 patients with peritonsillar abscess, operated on at various stages, only slight local reaction occurred, and no glandular swelling or severe pain was observed after the operation. With one exception, a drop in temperature occurred soon after the procedure. These results compared favorably with those of tonsillectomies performed for chronic recurrent tonsillitis and of operations done two fever-free days after acute tonsillitis. The authors, therefore, conclude that not only is tonsillectomy for peritonsillar abscess relatively free from danger but also it is the procedure of choice.

Tonndorf [122] presented a group of 542 patients with peritonsillar abscess who were treated by simple incision through the anterior pillar, with perfect results in 99 per cent. Cases totaling several thousand are cited from other clinics, which are well satisfied with this technic. It is admitted that enucleation during the height of the abscess is not so serious as has generally been supposed, yet enucleation is unnecessary except in cases of sepsis and in the few cases in which the abscess fails to heal with simple incision. Enucleation is attended with greater morbidity than is incision, and one of its proponents has stated that it is contraindicated in the phlegmon stage before abscess formation.

After discussing briefly the origin, pathologic development and diagnosis of peritonsillar abscess, Linck [123] also considers the therapy of

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121. Hofer, G., and Motloch, T.: Tonsillectomy and Peritonsillar Abscess, Ztschr. f. Hals-, Nasen- u. Ohrenh. **35**:390, 1934.

122. Tonndorf: Tonsillectomy During the Acute Stage of Peritonsillar Abcess, Ztschr. f. Hals-, Nasen- u. Ohrenh. **36**:316, 1934.

123. Linck, A.: Simple Incision, Incision and Secondary Tonsillectomy and Tonsillectomy in the Course of Peritonsillar Abscess, Ztschr. f. Laryng., Rhin., Otol. **25**:79, 1934.

this condition. The basis for therapy consists in the discovery and emptying of every purulent peritonsillar inflammation. Healing should occur quickly and surely without leaving an abscess focus, and care should be used that not only the peritonsillar abscess but also the chronically inflamed tonsils are radically removed. Three types of operative treatment of entirely different value are considered. Simple incision is the oldest type and the one most commonly employed. The advantage of this is its ease of application either by a general practitioner or in office practice. The disadvantages are well known, especially the difficulty of completely emptying the abscess. Incision followed by tonsillectomy connects the treatment of the abscess with secondary prophylaxis. The advantage of this method lies in the fact that by removing the tonsils a number of possible complications are avoided which might originate from the diseased tonsils. The disadvantages are numerous and include those which apply to incision alone. Two operative procedures are necessitated, and it is impossible to say just what is the best time for the second. Finally, the author considers immediate tonsillectomy in cases of abscess as both a curative and a prophylactic treatment. The indication for this is a purulent peritonsillar phlegmon or inflammation at any stage of development or repair. The advantages are obvious. There is no purulent peritonsillar inflammation which cannot be treated by this means. No abscess cavity exists after tonsillectomy. Disadvantages which have been advanced by opponents of this treatment include the danger of operating on acutely inflamed tissue, the special burden on severely ill patients and the risk for those with high blood pressure. The author is inclined to minimize these claims. He believes that tonsillectomy serves as a great step forward in the treatment of peritonsillar abscesses.

Zöllner[124] attempts to answer several questions in regard to the treatment of peritonsillar abscess. The first question, whether tonsillectomy is safe in the acute stage, he answers by reviewing 91 cases in which tonsillectomy was performed during this stage. In 4 instances, in spite of relatively good preoperative conditions, fatal septicemia resulted. By further analysis he concludes that although tonsillectomy in the acute inflammatory stage is well borne in general, there are individual cases in which it may lead to severe complications. In regard to the question whether simple incision suffices for the healing of the acute inflammatory process, the author states that about two thirds of the patients treated in this way rapidly become free from fever. The final question asked is whether there are satisfactory indi-

124. Zöllner, F.: Tonsillectomy or Incision in Peritonsillar Abscess? Ztschr. f. Hals-, Nasen- u. Ohrenh. **35**:509, 1934.

cations for tonsillectomy. As a result of a critical analysis of the clinical and histologic evidence available, Zöllner feels that there are three points which serve as indications for tonsillectomy: (1) delayed involution of the local and general signs after simple incision, (2) failure of the incision to elicit pus, for example, hidden abscess or phlegmon, and (3) septic states resulting from the tonsillar condition and also absence of demonstrable peritonsillar inflammatory signs.

Canuyt [125] describes the method which he has advocated for opening a peritonsillar phlegmon. At the termination of the operation, the upper pole of the tonsil is partially dislocated, so that the first stage of a tonsillectomy has already been achieved. Tonsillectomy is carried out in one of three stages: (1) during acute inflammation, (2) in the subacute stage and (3) in the quiescent stage. Although Canuyt has carried out many tonsillectomies during the acute stage, indeed, his knowledge of the pathologic anatomy of peritonsillar phlegmons has largely been derived from such treatment, he does not recommend this method, which he considers unnecessarily painful, dangerous and often incomplete. On the other hand, if the affected tonsil is allowed to settle down for a period of weeks or months, as is the usual custom, it will reattach itself to the tonsillar fossa and the fibrous adhesions will render the operation difficult. Moreover, one runs the risk of the patient's putting off the evil day, and this is one of the reasons why so many recurrent quinsies are seen. The author advocates operating in the "warm" stage as opposed to the "hot" and "cold" stages. The removal of the tonsil is carried out four or five days after the evacuation of the phlegmon by his method, that is to say, when the temperature has dropped to nearly normal. At about this time, the edema has disappeared, the infiltrated tissues have regained their mobility, the fossa is wide open, the walls of the abscess cavity are well defined and a substantial portion of the tonsil is freed. The operation is easy, painless and safe. As usual, local anesthesia is advocated.

Tato [126] has collected a total of 356 cases of peritonsillar abscess treated by tonsillectomy in recent years, including 29 of his own. In a single case was there a septic complication with signs of rheumatism, and in a case of the author's a hemorrhage occurred, which was easily arrested. In 3 of the 29 cases, the abscess was bilateral, so that he removed 32 tonsils altogether in the treatment of the abscesses. The results have been excellent, providing a safe and quick cure of the abscess and eliminating relapses and late recurrences with their

125. Canuyt, G.: Total Tonsillectomy in the "Sub-Acute" Stage After a Wide Incision of a Peritonsillar Abscess, Ann. d'oto-laryng., February 1934, p. 130.

126. Tato, J. M.: The Treatment of Quinsy and Peritonsillar Abscess by Tonsillectomy as the Method of Choice, Rev. espafi. y am. de laryng. **24**:12, 1933.

possible complications. In the series the extremes of age were 4 and 70 years. The author employs local anesthesia. He gives the clinical histories of his own patients and a full bibliography.

Although physicians are all familiar with phlegmonous collections of the upper pole of the tonsil, those occurring in connection with the inferior pole are little known. Escat [127] has come across only 3 cases of this condition, and these are reported in detail. There are certain features of this condition which should be emphasized: 1. The initial dysphagia, which is localized to the lower tonsillar pole, is markedly severe; pain is referred to the ear, and sleep becomes impossible. 2. The swelling is strictly limited to the lower pole of the tonsil between the fold of His and the lower third of the anterior pillar. 3. There is a definite tendency for the inflammatory edema to spread to the laryngeal region. Indeed, one may ask oneself whether in some of those obscure cases of acute edema of the larynx arising from some unknown cause the condition may not have originated in this fashion. 4. Pyrexia is not a feature. 5. The evolution of the abscess is rapid, so that evacuation may result in about three days. 6. Diagnosis is facilitated by digital pressure.

The treatment of this condition is surgical. The abscess is opened either with the electrocautery knife, as recommended by Moure, or with the sharp pointed hook, as preferred by Escat. His method of operation is shown in an illustration.

After presenting two full clinical records of cases which terminated, one fatally and the other with recovery, Lapouge [128] sums up his conclusions as follows:

> A gangrenous phlegmon of the tonsillar fossa should be clearly distinguished from the more common peritonsillar abscess. It occurs in debilitated subjects, particularly in those with a history of renal disease. The onset is sudden. The morbid entity is established in a few hours, and the usual preliminary stages are absent. Indeed, abscess formation appears to be the primary stage. Dysphagia is violent, and unilateral edema and glandular reaction are marked. There is a high temperature and rigor. Hematuria and slight albuminuria are often present. The pus is characteristic: as soon as it is evacuated, it appears dark brown and has a revolting odor. The edges of the abscess cavity are gangrenous. In both the cases reported, hemoculture gave negative results, so that there was no definite septicemia, but the patient's general condition indicated a high degree of toxemia. Treatment should be carried out as soon as possible, and the opening into the abscess should be as wide as possible. Treatment of the general condition is most important, and the injections of propidon are regarded as essential.

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127. Escat, E.: Inferior Peritonsillar Abscess, Ann. d'oto-laryng., October 1933, p. 1137.

128. Lapouge: Gangrenous Phlegmons of the Tonsillar Fossa, Ann. d'oto-laryng., June 1935, p. 643.

Bilateral encapsulated peritonsillar abscess is a rare condition in children. Teed [129] reports a case of this type and gives a comprehensive review of the literature. He states that it would be interesting to speculate on the incidence of peritonsillar abscess in young children in view of the rather common occurrence of retropharyngeal infection. A survey of the literature, however, reveals no reliable opinion on the matter, and no evidence is presented on which to base a theory. A similar case is reported by Sugaya.[130]

The patient was an infant aged 14 months, with a high temperature and difficulty in swallowing. The right tonsil and the epipharynx showed redness and swelling. On digital palpation a profuse thick collection of pus ran out of the right external auditory meatus. After this manipulation had been repeated several times, the local swelling gradually subsided, and recovery took place in two weeks.

According to Rosenberg and Berke,[131] retropharyngeal abscess is considered to be a serious condition, requiring fine judgment for proper treatment. The frequent occurrence of sudden death from incision or spontaneous rupture of the abscess is mentioned. The frequency of severe hemorrhage, thrombosis or rupture through the external auditory canal is discussed. Several cases are reported. Fluctuation should preferably be noted before the abscess is incised.

The grave complication of hemorrhage from pharyngeal and peritonsillar abscesses is not so infrequent as might be supposed, as numerous isolated case reports have been published. A comprehensive review of the subject was made by Salinger and Pearlman,[132] who report 10 cases personally observed (6 with fatal termination) and give a detailed analysis of 231 cases recorded in the literature. Abnormal tortuosity of the internal carotid artery is not uncommon. True carotid aneurysm is rare, but false aneurysm, the result of septic infection of the arterial wall, is a frequent cause of hemorrhage from an abscess of the pharynx. An analysis of the results appears to illustrate the advantage of ligation of the carotid artery. Although ligation of the common carotid artery is frequently followed by serious cranial complications due to the sudden anemia of the brain, this is a risk which must be

129. Teed, R. W.: Bilateral Encapsulated Peritonsillar Abscess in a Child: Review of the Literature and Report of a Case, Arch. Otolaryng. **22**:90 (July) 1935.

130. Sugaya, S.: Peritonsillar Abscess Following an Interesting Course, Otorino-laryng. ital. **8**:437, 1935.

131. Rosenberg, L., and Berke, M.: Complications of Retropharyngeal Abscess, J. Pediat. **4**:764, 1934.

132. Salinger, S., and Pearlman, S. J.: Hemorrhage from Pharyngeal and Peritonsillar Abscesses: Report of Cases, Résumé of Literature and Discussion of Ligation of Carotid Artery, Arch. Otolaryng. **18**:464 (Oct.) 1933.

faced when one is confronted with a serious hemorrhage, and the possible advantages outweigh the disadvantages. A detailed account of the recorded cases is given in tabular form.

A case of serous meningitis of allergic nature is reported by Sheldon [133] in which an attack of acute tonsillitis was followed after an interval of eight days by a generalized scarlatiniform rash and by two groups of symptoms which developed concurrently: (1) a series of involvements of the larger joints, characterized by pain and effusion, the latter being especially prominent in the knees, and (2) serous meningitis of sufficient severity to give rise to 4 diopters of papilledema. Both groups of symptoms subsided after the development of a peritoneal abscess caused by a hemolytic streptococcus; its drainage led to complete recovery. The diagnosis of hydrocephalus caused by serous meningitis was established by the normal character of the cerebrospinal fluid, its increased pressure and the subsidence of symptoms, including the papilledema, after two lumbar punctures. The nature of the effusion into the joints was not established by the puncture of a joint, but its complete subsidence indicated that the fluid was certainly not purulent, and it was therefore assumed to have been serous.

According to Gray,[134] streptococcic meningitis is usually a fatal disease, with but 66 reported instances of recovery over a period of thirty-four years. Treatment in most cases in which recovery has occurred has been more conservative than the generally published regimen and consists principally of eradication of the initial focus, spinal taps, intraspinal and intravenous injections of serums, and blood transfusions.

In a case reported by Gray treatment was conservative, and it would be difficult to state the definite cause of recovery. It is the opinion, however, that maintaining the physiologic balance as nearly as possible had more to do with recovery than any other procedure. In 8 cases the cause was tonsillitis. In practically all others the meningitis was due to otitis media and mastoiditis.

Of 354 patients with tonsillitis, de Wesselow and his associates [135] found that 14.4 per cent showed definite early albuminuria and 6.2 per cent late albuminuria. From the urinary and clinical observations it would seem probable that focal nephritis occurred in 35 (10 per cent) and diffuse glomerulonephritis in at least 3 (0.8 per cent). No correlation was found between the predominance of hemolytic streptococci in the throat at the time of the acute infection and the incidence of focal

133. Sheldon, J. H.: Serous Meningitis of Allergic Nature, Lancet **1**:798, 1933.

134. Gray, H. J.: Streptococcic Meningitis: Report of Case with Recovery, J. A. M. A. **105**:92 (July 13) 1935.

135. de Wesselow, O.; Goadby, H., and Derry, D.: Tonsillitis and Albuminuria, Brit. M. J. **1**:1065, 1935.

nephritis. Seasonal variations of external temperature did not appreciably affect the incidence of early or late albuminuria. The incidence of predominant hemolytic streptococcic infection in the throat was not greater in those patients in the general series who afterward complained of rheumatic pains.

Hays [136] reports an interesting but simple case which brings up a number of problems of vital interest. A tonsillectomy was performed on a patient whose coagulation and bleeding time were prolonged to seven minutes. Three hours after operation a severe hemorrhage occurred. After transfusion the bleeding ceased immediately. Two days later he acquired acute gangrenous appendicitis. The appendix was removed, and eventually he recoverd.

In an article entitled "Some Considerations of Adenoid Bleeding" Richards [137] presents a comprehensive survey of the data obtained from the observation of a large group of patients. Statistical tables point out the incidence of operative bleeding of the tonsils and adenoids as noted by a number of observers in different clinics and hospitals. The methods or technic of operating were also taken into consideration. The relationship between loss of blood, bleeding from adenoids and postoperative bleeding was also investigated. There was a direct correlation between the size of the adenoid and the immediate loss of blood attending its removal. More bleeding was noted in instances in which the time of operation was prolonged. Ten cases in which there was a greatly increased general bleeding time were checked in respect to the duration of adenoids bleeding and the tendency to postoperative bleeding. The average bleeding time for these 10 was seven and three-tenths minutes, against an average for the whole group of two and five-tenths minutes. Yet these patients showed an average adenoid bleeding time of six and three-tenths minutes, five being the group average. Among these 10 cases there occurred only 1 instance of postoperative bleeding requiring packing. The point was brought out that postoperative bleeding is essentially independent of a prolonged general and immediate loss of blood and an excessively large adenoid. Richards, however, believes that the depth of anesthesia plays an important part in postoperative bleeding. The question of anesthesia in respect to tonsillectomy and adenoidectomy was therefore discussed in detail. The method of removing the adenoid was also considered as having some bearing on the liability to subsequent hemorrhage. Richards finally concludes that the best results and the fewest complications come from a careful preoperative investigation of the patient, the employment of

136. Hays, H.: Tonsillectomy on a Bleeder; Transfusion; Acute Gangrenous Appendicitis, Peritonitis, Recovery, Laryngoscope **44**:315, 1934.

137. Richards, L.: Some Considerations of Adenoid Bleeding, Ann. Otol., Rhin. & Laryng. **44**:117, 1935.

a competent anesthetist, who knows at all times the depth of the anesthesia, a careful technic carried out with neither haste nor delay and an assurance of returning pharyngeal reflexes before removal of the adenoid.

Rubenstein [138] reports a case and reviews the literature on the subject of surgical emphysema of the head, neck and chest complicating tonsillectomy. The evidence presented suggests that in this case the entrance of air was through the tonsillar bed. Cough during the operation was assumed to have brought about the injury to the inner fascial covering of the superior constrictor, although the capsular surface appeared to be intact.

According to MacCready,[139] subcutaneous emphysema following tonsillectomy is not uncommon, although relatively few reports of its occurrence have appeared in the literature. He states that it is not a serious complication but is likely to be disconcerting to the surgeon and that its prevention should be possible in most instances. Two cases which have occurred in MacCready's experience were reported. One of the patients was operated on under local anesthesia and the other under general anesthesia. The patient who was operated on under local anesthesia had a terrific spasm of coughing, and the second patient, operated on under general anesthesia, had increased intrathoracic pressure which developed during a spasm in which she clamped her teeth together, so that a tremendous effort was required in order to breathe. MacCready emphasizes the importance of preventing anything which would produce explosive respiratory effort and give rise to increased intrathoracic pressure.

Lowenthal [140] conducted a postoperative bronchoscopic study of 21 patients with the idea of determining the tracheobronchial aspiration of buccopharyngeal secretion during ether anesthesia. It was found that in operations on parts which do not drain into the pharynx with the patient in the horizontal position, aspiration of the pharyngeal contents occurred in 76 per cent of the cases. When aspiration does take place, it is likely to involve the entire bronchoscopically visible portion of the tracheobronchial tree. Aspiration in patients subjected to tonsillectomy was twice as frequent when ether anesthesia was used as when local anesthesia was used.

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138. Rubenstein, C. L.: Surgical Emphysema of the Head, Neck and Chest Complicating Tonsillectomy and a Review of the Literature, Ann. Otol., Rhin. & Laryng. **44**:509, 1935.

139. MacCready, P. B.: Subcutaneous Emphysema Following Tonsillectomy: Report of Two Cases, Arch. Otolaryng. **22**:331 (Sept.) 1935.

140. Lowenthal, G.: Tracheobronchial Aspiration of Buccopharyngeal Secretion During Ether Anesthesia: Immediate Postoperative Bronchoscopic Study of Twenty-One Patients, Arch. Otolaryng. **21**:561 (May) 1935.

Acute pulmonary infections after tonsillectomy are not serious as a rule and clear up rapidly. As regards their cause, Schütz [141] considers three possibilities: (1) aspiration of blood into the bronchi, (2) spreading infection via the lymphatics and (3) infection of the blood stream by means of emboli. The aspiration theory is almost given up now. In animals it is practically impossible to produce a pulmonary abscess by introducing blood and pus into the bronchial tubes. On the other hand, injection of infected material into the jugular vein at once produces a septic infarct in the lung.

In the clinical cases reported, the symptoms began usually in the first twenty-four hours after operation, and signs indicating small bronchopneumonic foci were present almost immediately. Schütz implies that such infections are clearly embolic in origin. At the same time he admits that the condition may be simply an acute flare-up of a mild bronchial or bronchopneumonic infection which is already present at the time of operation, especially at periods when "colds" are prevalent.

As is usual with German writers, Schütz attributes the large number of pulmonary complications mentioned in the American literature to the use of general anesthesia. In German clinics tonsillectomies are mostly done with the area under local anesthesia or after a very short inhalation of ethyl chloride.

Ranson and McGolrick [142] report a case of a pulmonary abscess following tonsillectomy, with operation and recovery. A brief discussion of the mode of production of such abscesses and their treatment follows.

Waldapfel [143] after a sojourn in the United States is unable to explain the relative infrequence of abscesses of the lungs after tonsillectomy reported in Germany. He thinks that it may be due to the superficial narcosis, with persistence of pharyngeal reflexes and to the fact that the operation is performed with the patient in the sitting position. When patients showing posttonsillectomy fever were investigated, however, he found eight in a relatively short time with various pulmonary complications which had been absent before the operation. In making the diagnosis, roentgenography was the most important factor. In all these cases the prognosis was good. The prophylaxis of pulmonary complications of tonsillectomy consists in several measures. Narcosis should be as light as possible, so that the pharyngeal and cough reflexes are maintained. The operation should be as short as

141. Schütz, W.: The Various Forms of Pulmonary Complications After Tonsillectomy, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **139**:369, 1935.

142. Ranson, F. T., and McGolrick, L.: Lung Abscess After Tonsillectomy, Brit. M. J. **2**:1020, 1933.

143. Waldapfel, R.: Pulmonary Complications After Tonsillectomy, Monatschr. f. Ohrenh. **68**:143, 1933.

possible. To reduce the infection of the blood stream, all possible asepsis of the oral cavity should be maintained. Waldapfel believes that the frequency of posttonsillectomy pulmonary complications in Germany is much greater than it was heretofore considered to be and that all patients showing fever after operation should be investigated with this in mind.

Guerard and his associates [144] report the case of a girl aged 9 years, who came to the hospital complaining of a red and painful swelling, which had appeared two days before in the left nasal ala and had spread rapidly to the left eyelid and then to the entire left side of the face. The temperature rose to 104 and 105 F.; the pulse was feeble, and the patient was stuporous. A few days later the upper eyelid on the left side suppurated and was incised, after which the skin of the entire lid sloughed away. Soon the left side of the scalp and face began to suppurate profusely and became denuded of skin. The septicema last eight months, with slow improvement. During this period osteo-arthritis of a finger of the right hand occurred, with extensive osteoperiosteitis of the right femur, osteitis of the right humerus, an abscess of the left lung and a renal complication, the presence of which was revealed by pyuria and hematuria. An unusual complication consisted in an inability to rotate the head, which developed during several days and was characterized by fixity of the head and neck, with massive cellulitis in the latter region, all depending on rotation of the atlas by which one side, usually the left, projected forward beyond the other and on a cervical curvature of the spine, with the convexity to the right. In this right-sided protrusion the spinous process of the axis could be felt, pushed to the right by the sliding forward of the axis and its rotation to the left. This was brought out by roentgenograms. The condition was diagnosed as nasopharyngeal torticollis. This disease was first described by P. Grisel in 1930 and has received the name "Grisel's disease." It occurs chiefly in girls from 8 to 12 years of age, following the appearance of an abscess in the retropharyngeal region or other angina of the throat. If it is not recognized and treated, it becomes chronic. In this stage a characteristic sign is the "sternal parados," in which the sternocleidomastoid muscle which contracts tonically is not that of the affected but that of the opposite side.

144. Guerard, J.; Potvin, R., and Jobin, J. B.: A Case of Nasopharyngeal Torticollis, Bull. Soc. méd. d. hôp. Universitaires de Québec **34**:58, 1933.

Abstracts from Current Literature

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EXPERIMENTAL RESEARCHES ON THE ORIGIN OF THE ACOUSTIC GANGLION IN AMPHIBIAN EMBRYOS. ERNEST VAN CAMPENHOUT, J. Exper. Zoöl. **72**:175 (Nov. 5) 1935.

In the amphibians Rana pipiens and Amblystoma jeffersonianum, unilateral extirpation of the otic placode, unilateral extirpation of the neural crest of the hyoid arch, extirpation of the rhombencephalon with the neural crest or heterotopic transplantation of the placode was performed on embryos from the stage of the wide-open neural fold (Amblystoma) to the stage of the beginning tail bud (frog).

The following results were obtained: absence of the acoustic ganglion after extirpation of the otic placode; presence of a normal ear with a normal acoustic ganglion and nerve after extirpation of the neural crest; formation of an acoustic ganglion without the acoustic nerve after removal of the rhombencephalon and adjacent neural crest; migration of acoustic ganglion cells from the otic epithelium, and formation of the ear vesicle with acoustic ganglia from heterotopic grafts.

These observations in amphibians were in complete accord with the results of previous work on the development of the acoustic ganglion in pig embryos. Since the work demonstrates that in two widely separated groups, amphibians and mammals, the acoustic ganglion cells are derived from the otic epithelium, without contribution from the neural crest, it is suggested that this origin of the acoustic ganglion holds for the entire vertebrate series.

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VASCULAR FORMS OF OCULAR PARALYSIS IN THE COURSE OF OTITIS AND MASTOIDITIS. J. REBATTU and A. COLRAT, Rev. d'oto-neuro-opht. **13**:565 (Sept.-Oct.) 1935.

Infection from the tympanum often reaches the point of the petrous process, and it is through this region that paralysis is produced. The third cranial, or oculomotor, and the fourth cranial, or patheticus, nerve traverse a triangular space between the attachments of the tentorium, perforate the dura and enter the external wall of the cavernous sinus; hence, they are not in close relation with the point of the petrous process. The structure of the petrous bone is variable, but, in general, the compact block formed by the labyrinth is surrounded by air cavities which communicate with the cavities of the middle ear and extend to the point. The point contains relatively large cells. In almost half the bones the structure of the point is not of the same type as that of the mastoid, but it is the same on the two sides. The abducens nerve in the posterior fossa is in relation with the middle cerebellar and internal carotid arteries and penetrates the dura just before reaching the petrous bone; the dural opening is from 3 to 4 mm. below the edge of the bone. The two dural openings are from 2 to 2.5 cm. apart. The nerve is then in immediate relation with the petrous apex and also with the inferior petrosal sinus. It then enters Dorello's canal, in which the nerve may be easily compressed by any increase in volume of the inferior petrosal or of the cavernous sinus. After leaving Dorello's canal, the nerve is in relation with the carotid artery and then penetrates the cavernous sinus. The trigeminus nerve perforates the dura at the superior edge of the petrous process, at which point it is in relation with the superior petrosal sinus. The gasserian ganglion lies on the anterosuperior face of the petrous process; directly beneath Meckel's fossa the bone, when pneumatized, contains more or less well developed cells. If the cells are not developed, Meckel's

fossa is in direct relation with the carotid canal. The third and fourth nerves lie to the inside of the point of the petrous process and above and medial to the sixth cranial nerve. Since they are not in direct relation with the process, their involvement in the course of otitis media and mastoiditis indicates a serious complication (extradural abscess, brain abscess or thrombophlebitis).

The syndrome of Gradenigo and Lannois is seldom encountered before the age of 5 years and is rare after 40, being most frequent between the ages of 5 and 15. It is more frequent in males. There is nothing pathognomonic about the otitis. Arrest of the discharge coincident with the onset of trigeminal neuralgia speaks in favor of petrositis. If the mastoid has already been opened, lack of correlation between the amount of discharge from the tympanum and from the mastoidectomy wound is significant. Pain in the trigeminal area occurs usually at an interval after the pain in the ear has subsided; it varies in character and may be located in the frontal, temporal, parietal, occipital, orbital or dental region. Dental pains are said to indicate a particularly grave prognosis. Usually there is no modification of sensibility. The most characteristic feature of the triad of Gradenigo is paralysis of the abducens nerve. The paralysis develops rapidly and is almost always homolateral, but may be heterolateral or bilateral. In either of the last two cases it may, in general, be assumed that the paralysis is due to hypertension or, rarely, to propagation of the infectious process from one petrous bone to the other. The temperature, the general condition and the cerebrospinal fluid are usually not modified, and facial paralysis is relatively frequent. Data obtained from roentgenography must agree with the clinical findings to be of value. The evolution is ordinarily favorable; recovery may occur without surgical intervention, but usually mastoidectomy is indicated. The syndrome of Gradenigo may be caused by phlebitis of the inferior petrosal sinus, propagation of the infection by way of the caroticotympanic canal or infection of the preformed cells in the tip of the petrosa. A benign and a severe form may be distinguished. In the former type there exist simply petrous cellulitis and meningeal congestion or, perhaps, toxicoinfectious neuritis, and recovery follows drainage of the mastoid cells. In the severe form the apical lesion is more serious—osteitis with extradural abscess.

Isolated paralysis of the sixth nerve may be classified as mild, moderate or severe. Acute otitis or exacerbation of a chronic suppuration is always present; a meningeal reaction may or may not be present. Only complete clinical and neurologic study can give information as to the causative lesion. In the mild form paracentesis of the drum membrane or exenteration of the mastoid is sufficient to cause the paralysis to disappear; probably the mechanism of the paralysis is venous congestion, which recedes with drainage. The rapid evolution and the absence of trigeminal neuralgia distinguish the paralysis from Gradenigo's syndrome. Meningeal signs, when present, are probably due to serous meningitis. In the moderately severe form the lesion is located in the petrous tip. The prognosis and evolution are the same as those of Gradenigo's syndrome. The prognosis is unfavorable in the severe form. Abscess of the cerebellum was the causal agent in sixteen of eighty cases of the severe form. The sixth nerve appears to be more sensitive to hypertension than to infectious processes, but Baldenweck collected seven cases of meningitis accompanied by paralysis of the sixth pair.

Isolated paralysis of the oculomotor (third) nerve has been observed in association with thrombophlebitis of the cavernous sinus and in suppurative meningitis. Isolated paralysis of the patheticus nerve in association with otitis media is exceptional. Lapersonne reported such a case and concluded from the associated labyrinthitis and the rapid recovery that the paralysis was of reflex nature. Diagnosis is difficult, and caution is necessary in arriving at a conclusion.

Multiple paralyses of the cranial nerves almost always terminate in death. When there is paralysis of the third and sixth nerves thrombosis of the cavernous sinus is frequently the cause, although paralysis of these nerves has been observed in cases of extradural abscess near the petrous tip. Multiple paralyses may accompany suppurative otogenous meningitis or brain abscess. Paralysis of this type arising from thrombophlebitis of the cavernous sinus is often a complication of

old otorrhea; either osteitis of the petrous tip determines an extradural abscess and thrombophlebitis by direct extension or, most frequently, there is extension of phlebitis from the lateral sinus by way of the superior or the inferior petrosal sinus or the carotid sinus.

Treatment is discussed at length. Extensive exenteration of the mastoid in cases of infection of the petrous tip is indicated; if the symptoms are not rapidly relieved, the tip must be opened, preferably by the superpetrous route.

DENNIS, San Diego, Calif. [ARCH. NEUROL. & PSYCHIAT.]

HISTOPATHOLOGIC ALTERATIONS OF THE EAR IN EXPERIMENTAL HYPERCHOLESTEREMIA. F. CARNEVALE-RICCI, Arch. ital. di otol. **47**:825 (Dec.) 1935.

Carnevale-Ricci states that the most important histologic alterations produced on the auditory apparatus of rabbits by experimental hypercholesteremia of long duration (between six and eight months) are those caused by extensive and grave fatty degeneration and lipoid infiltration of all tissues and structures of the auditory apparatus, especially of the nerve tissues. The acoustic nerve and its branches are almost entirely involved in the process. Next in involvement are the cochlear structures, the vessels and the muscular, cartilaginous and bony tissues. The changes as a whole correspond to those seen in cadavers of persons who during life suffered from hypercholesteremia. The arterial lesions do not produce the typical picture of arteriosclerosis in man.

EDITOR'S ABSTRACT.

INFLUENCE OF CONGENITAL SYPHILIS ON THE OSSIFICATION OF THE LABYRINTHINE CAPSULE IN THE NEW-BORN. W. DÖDERLEIN, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **140**:229, 1936.

Döderlein describes histologic observations on the petrosas of newly born infants with congenital syphilis. Since the changes appeared only in the zone of enchondral ossification of the labyrinthine capsule, he reports only the observations on this region. In some cases the conditions were normal, but in others he observed an unusual acceleration of the ossification of this zone. In the latter group, the bone at many sites was even more compact than that in the labrinthine capsule of adults. The author thinks that this acceleration in the ossification of the labyrinthine capsule is caused by syphilis. Thus it has been proved that congenital syphilis may cause not only retardation but acceleration of the ossification of the labyrinthine capsule. Whether these pathologic processes of ossification may later result in auditory disturbances cannot be decided as yet. The nervous elements of the cochlea were normal in the instances of pathologic ossification.

EDITOR'S ABSTRACT.

HISTOLOGIC OBSERVATIONS TWO YEARS AFTER PARTIAL RESECTION OF THE LABYRINTH. W. STUPKA, Monatschr. f. Ohrenh. **69**:1281 (Nov.) 1935.

Stupka thinks that it is of interest to know how the closure toward the brain takes place after total labyrinthectomy and also what are the final results after partial operation on the labyrinth, for instance, after an operation involving only the semicircular canals and the vestibule, leaving the cochlea unimpaired. The operation in the case described here belongs to the latter type. Almost two years elapsed between the operation on the labyrinth and the death of the patient. During that time the patient had been repeatedly examined. The surgical cavity in the labyrinth had healed and had become completely covered with epidermis. The first operation had not arrested the tumor-like growth of the cholesteatoma. It apparently developed further during the after-treatment, the course being entirely afebrile and without subjective and objective vestibular symptoms. The cholesteatoma entered the labyrinth on two sites (in the region of the ampulla of the horizontal semicircular canal and in the region of the fenestra vestibuli). In describing the histologic aspects Stupka emphasizes as especially noteworthy the complete collapse of the ductus cochlearis, with complete atrophy of the organ

of Corti and of the membrana tectoria, and the severe atrophy of the ligamentum spirale cochleae and of the lamina spiralis ossea. The author is convinced that in spite of the histologic changes which were detected in this case a partial operation was justified. The preservation of the cochlea was justified not only in view of the possibility of a restoration of partly abolished functions but also because it was free from invasion by the cholesteatoma.

EDITOR'S ABSTRACT.

ABILITY OF PATIENTS WITH DISEASE OF THE EAR TO DIFFERENTIATE INTENSITIES OF SOUND EXPERIMENTS WITH THE OTO-AUDION. W. BRINITZER, Monatschr. f. Ohrenh. **69**:1301 (Nov.) 1935.

Brinitzer investigated the ability of patients with disease of the ear to differentiate intensities of sound, and he compared this ability with the capacity for hearing. The studies revealed that the reduction of the threshold values does not parallel the reduction of the ability to differentiate intensities. It seems that the reduction in the capacity for the differentiation of intensities of sound is determined by the type of the auditory defect. Patients with acute or chronic otitis media, with the exception of those who underwent a radical operation and of those with severe inflammatory processes, had a good capacity for differentiation of intensities of sound. For the patients with defects of the internal ear whose anamnesis revealed a cranial trauma, the capacity for differentiation of intensities of sound was usually greatly reduced. The same was true of patients whose internal ear had been impaired by noises. For the patients with endogenous hardness of hearing in the internal ear the results of the tests for differentiation of intensities lacked uniformity, as was the case in patients with otosclerosis.

EDITOR'S ABSTRACT.

FORMATION OF NEW BONE TISSUE IN COURSE OF MASTOIDITIS. E. ZIEGLER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:159, 1936.

Ziegler says that the fact that new bone tissue forms in mastoiditis is well known. However, so far there exists no systematic description of this process from its beginning to the development of mature bone tissue that no longer undergoes essential changes; so the author gives his attention to this problem. After discussing the periosteal formation of new bone during normal growth, he gives his attention to the inflammatory formation of new bone. He describes the formation of osteoblasts and the formation of new bone by means of primary ossification and by the development of osteoblastic coverings. Further, he discusses the spongiform development of new bone and finally the seamlike formation of new bone. After this, he takes up the question as to what becomes of the newly formed bone. He gained the impression that the newly formed seams remain generally undisturbed. At longer intervals new seams are added, without changes appearing in the older or younger seams. The spongiform formation of new bone at first presents a rather widely spaced framework of small trabeculae by which cavities and air and marrow spaces are reduced. It is always the surface of the spongiform bone directed toward the lumen which is first changed into compact bone and into a bony capsule that is covered with endosteum. Later the spongiform appearance disappears also in the portions that are closer to the old bone. Finally, a capsule is no longer recognizable because the entire bone has been changed into a compact mass. At first it can still be differentiated from the old bone by a different color, but finally this also disappears.

In discussing the significance of the formation of seamlike and of spongiform bone the author says that between small and wide seams and between regular and irregular, dented and bayed seams up to spongiform formations, there appear all transitional forms. If the formation of new bone is regarded as a part of the defense mechanism against the inflammation, it may be assumed that in case of slight inflammation and of small cavities the seams will be capable of shutting off the process and of strengthening the walls. However, if in case of severe inflammation considerable destruction takes place and large cavities form, mere

formation of a seam will not be adequate, and extensive spongiform formations will appear. The author further discusses the relationship between the formation of new bone and the age of the inflammatory processes and then discusses the possibility of a pneumatized mastoid becoming sclerotic as the result of the formation of new bone in the course of mastoiditis. He is unable to give a definite answer to the latter question, and although he does not absolutely deny the possibility, he considers it extremely rare and says that he has never observed it.

EDITOR'S ABSTRACT.

Pharynx

CYTOPLASMIC INCLUSION BODIES IN THE HUMAN THROAT. J. BROADHURST, R. M. LIMING and M. E. MACLEAN, J. Infect. Dis. **58**:134, 1936.

Cytoplasmic inclusion bodies may be demonstrated in the human throat and other parts of the upper respiratory area. A relatively high proportion of persons with certain of the milder respiratory irritations carry these inclusion bodies. The carriers may show an increasingly heavy incidence in the epithelial cells of the throat—both as to the number of cells affected and as to the number of bodies per cell. The inclusion bodies are rarely present in epithelial cells which contain bacteria. They are easily demonstrated in the epithelial cells of affected persons, but they do not persist, in the same form at least, outside the host cells.

The epithelial inclusions seem acceptably classified as inclusion bodies: They multiply only when living tissue is available. They stain pink with Giemsa's stain (after fixation in Zenker's fluid). An aggregate structure (as in the Negri bodies of rabies) and a capsule may be demonstrated by appropriate stains. Special stains have been developed for their ready differentiation from other cell parts and constituents and extraneous material: notably a nigrosine-sodium chloride preparation for throat specimens and a methyl green-pyronine stain for embryo chick cultures. Cytoplasmic inclusion bodies have been cultivated in throats of monkeys and in embryo chick tissues.

FROM THE AUTHORS' SUMMARY. [ARCH. PATH.]

THE VIGGO SCHMIDT TEST. P. A. SAINZ, G. PRADO and W. DOMÍNGUEZ, Med. de los niños **36**:289 (Oct.) 1935.

The Viggo Schmidt test consists of a study of the neutrophilic response in the blood count following infection and massage of the tonsils. Two counts are made early in the morning; then digital massage for each tonsil for two minutes is carried out. Leukocyte counts are made every five minutes for half an hour. In normal tonsils there is a decrease in the total number of leukocytes, especially of the lymphocytes. If the tonsils are infected, the massaging produces leukocytosis and polynucleosis, with corresponding lymphopenia. This phenomenon is explained by the assumption that massage of the chronically infected tonsil liberates bacteria, toxins and products of the endolysis of polymorphonuclear neutrophils. When these enter the circulation, they have a positive chemotaxis for polymorphonuclear neutrophils, which produces the polynucleosis.

DWYER, Kansas City, Mo. [AM. J. DIS. CHILD.]

CLINICAL AND PATHOLOGIC ASPECTS OF TONSILLOTOMY. J. JESCHEK, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **140**:197, 1936.

Jeschek points out that although in the last three decades tonsillectomy has become more customary, tonsillotomy is still often resorted to in cases in which the tonsils are only hypertrophic. To be sure, it was early pointed out that tonsillotomy favors the development of tonsillitis and of inflammation of the cervical glands and that a large percentage of patients who underwent tonsillotomy were subject to rheumatic, polyarthritic and myocarditic processes. The author reports observations on twenty patients who underwent tonsillotomy about eight

years before examination. He found that all of them had a predisposition for tonsillitis and that thirteen had severe local or general complications. The tonsils of sixteen were subjected to histologic examination, and it was found that in nearly all of them there existed chronic atrophic tonsillitis, with severe cicatrization, cysts and inflammatory reactions. The examination of tonsils which were removed shortly after the tonsillotomy revealed that the changes which seem to be the causes of the frequent disorders in tonsillar stumps are the direct result of tonsillotomy. The favorable results of the total removal of the tonsillar remnants indicate that tonsillectomy is the best method. EDITOR'S ABSTRACT.

Larynx

THE LARYNGEAL DISEASE OF THE EMPEROR FREDERICK. L. COLLEDGE, J. Laryng. & Otol. **51**:31 (Jan.) 1936.

Colledge studied the illness of the Emperor Frederick from the pathologic standpoint in the light of present knowledge.

Crown Prince Frederick, who was under the care of Gerhardt, had been suffering from disease of the larynx since the autumn of 1886. A diagnosis of cancer had been made, and a thyrotomy was to be undertaken by von Bergmann, who had made all the necessary preparations. It had been decided, however, that before an operation of such gravity was performed another opinion should be sought. In consequence, Mackenzie was summoned to Berlin and was requested to examine the Crown Prince.

Mackenzie saw a growth about the size of a split pea on the posterior part of the left vocal cord, lying over the processus vocalis. On phonation part of the growth disappeared. Mackenzie stated that this showed that it was partly attached to the under-surface as well as to the side of the cord, or, in other words, was partly subglottic. He expressed the opinion that the diagnosis of cancer had been made on insufficient evidence and that a portion of the tumor should be removed for examination under the microscope. He performed this operation, and the piece removed was sent to Virchow. Virchow reported that the growth was benign. He called attention, however, to a cell nest present in the midst of proliferating epithelium and stated that the disorder might be pachyderma laryngis. Mackenzie, in consequence, advised removal of the growth with forceps, each portion removed to be sent to Virchow. He did not attempt it at the time, however, but left Berlin and did not return until June 7. On the following morning he again removed a piece of the growth. Virchow issued a report, in which he stated that the lesion was pachyderma verrucosum and that in no part could an ingrowth of this epithelial formation into the mucous membrane be detected. The squamous epithelial cells contained colloid, and cell nests were present.

On the strength of this report it was decided to treat the condition by endolaryngeal extirpation of the growth with forceps. The Crown Prince arrived in England to take part in the jubilee procession on June 21. On June 28 another piece, thought to be the entire growth, was removed and sent to Virchow, who again found no evidence of malignant changes in the tissue.

During October the swelling below the left cord increased in size and began to ulcerate, and by the end of the month another swelling had appeared below the right cord. Early in November the Crown Prince removed to San Remo, and the subglottic tumors began to increase rapidly. Schrötter was summoned from Vienna and made a diagnosis of cancer, with the recommendation that the entire larynx be excised or, as an alternative, that a palliative tracheotomy be performed. The patient declined excision of the larynx but agreed to submit to tracheotomy when necessary.

The left half of the larynx began to swell externally, and the growth extended rapidly inside the larynx. The obstruction which resulted was relieved by sloughing. Increasing dyspnea, however, had to be relieved by tracheotomy, which was performed by Bramann on February 9. A month later any doubt concerning the diagnosis was dispelled when Waldeyer found particles of cancerous tissue in the

sputum. The patient, now Emperor, was able to return to Berlin, but the disease progressed steadily, and a fatal termination ensued on June 15, 1888. A necropsy was performed the following day by Virchow and Waldeyer. It revealed destruction of the larynx by growth and perichondritis.

Two questions at once arise: First, why did Mackenzie fail to recognize the nature of the disease? Second, why did he fail to obtain specimens of tissue from which a correct diagnosis could have been made by Virchow? The author believes that the answer to both questions is that the cancer was subglottic with a small extension upward in the region of the vocal process and was not an ordinary cordal growth. If von Bergmann, although his diagnosis was correct from the first, had performed the operation which he proposed in May 1887, he would have found the growth far more extensive than he expected. It is likely that had the larynx been opened in May 1887, it would have been found when the interior was exposed to full view that the extent of the growth already called for a total extirpation of the larynx. In spite of the subsequent delay and the onset of perichondritis, the patient might still have been saved in November by total laryngectomy, as advised by Schrötter, but the operative risk would naturally have been greater. The attempt by Mackenzie to remove pieces with forceps by the indirect method, when the tumor was subglottic, merely resulted in misleading pathologic reports. To sum up, it seems that although Gerhardt and von Bergmann made a correct diagnosis of cancer, even they failed to appreciate fully the type and extent of the lesion.

EDITOR'S ABSTRACT.

Nose

THE COMMON COLD. A. R. DOCHEZ, K. C. MILLS and YALE KNEELAND JR., J. Exper. Med. **63**:559, 1936.

Detailed descriptions are given of the methods employed to isolate the virus of the common cold, to preserve and cultivate it and to test its activity in human volunteers. The virus can be isolated easily from properly selected patients and cultivated in a tissue medium. When kept in the original nasopharyngeal washings it will survive at icebox temperature under anaerobic conditions for at least thirteen days. If the nasopharyngeal washings are frozen and dried in vacuo, the virus retains its activity for at least four months. It has been proved to multiply in a medium containing chick embryo. Cultures in such a medium retain their capacity to produce the typical infection in human beings through many transfers involving a period of several months. Attempts to cultivate the virus have been successful in seven of eight instances. Prolonged cultivation in a tissue medium eventually leads to loss of activity. Strains under cultivation maintain their potency best when transfers are made at two and three day intervals. After removal from the incubator a culture of the virus rapidly becomes inactive whether it is kept under seal in the icebox or frozen and dried in vacuo. The destructive action of the medium can be prevented if the culture is mixed with acacia before freezing and drying in vacuo.

FROM THE AUTHORS' SUMMARY. [ARCH. OPHTH.]

EXPERIMENTAL INTRANASAL INFECTION WITH CERTAIN NEUROTROPIC VIRUSES: PREVENTION BY MEANS OF CHEMICALS INSTILLED INTO THE NOSTRILS. C. ARMSTRONG and W. T. HARRISON, Pub. Health Rep. **51**:203, 1936.

The instillation of various chemicals into the nostrils of mice and monkeys tends to prevent intranasal infection of the mice with the virus of the St. Louis type of encephalitis and of the monkeys with the virus of poliomyelitis. Trinitrophenol in concentrations of from 0.32 to 0.64 per cent, either alone or combined with alum, was found to be superior to 4 per cent alum and to be the most satisfactory and efficient experimental agent so far tried by the writers. Trinitrophenol in the concentration and amounts employed was devoid of detectable general or local

injurious effects on animals. Sixteen applications sprayed into the nostrils of the authors by means of an atomizer produced no detectable injurious effects. It is believed that trinitrophenol exerts its protective effects locally either by rendering the mucous membranes less permeable to infection or possibly by direct action on the virus or in both ways. Its use does not prevent the development of specific immunity in mice following a subsequent intranasal instillation of the virus of encephalitis. When it was given to mice one and two days before, one and two days after, or on the same day as the instillation of virus, it led to decreased susceptibility to the virus in all these mice as compared with nonprepared controls. The protection against intranasally inoculated poliomyelitis afforded by 0.32 per cent trinitrophenol is apparent for at least four to seven days after its last administration. Intranasally instilled chemicals effective in preventing encephalitis in mice have been found effective against poliomyelitis in monkeys, suggesting that such products may be utilized as an indicator in a further search for more effective prophylactic agents in the latter ailment.

FROM THE AUTHORS' SUMMARY. [ARCH. OPHTH.]

SYNDROME OF NASAL NERVE FROM BACILLARY TOXEMIA. C. CHARLIN, Ann. d'ocul. **173**:25 (Jan.) 1936.

Charlin states that he knows well the tuberculous toxemia with loss of weight, asthenia, loss of color and sometimes vesperal temperature which occurs without clinically observable pulmonary lesions. Fluoroscopy and better still roentgenography show thoracic pulmonary shadows, especially at the hilus. The condition is generally found among young adults, especially women. In many cases he has obtained remarkable cures with rest and tuberculin therapy. He has often been consulted for blepharitis, for blepharokeratitis and particularly by adults for iritis and cyclitis. He observed a syndrome of the nasal nerve in two patients who, to his great surprise, both gave positive reactions to the Mantoux test. He recollects that nasal neuritis gives a syndrome characterized by (*a*) symptoms in the anterior pole of the eye (ulcerative keratitis, iritis), (*b*) symptoms of acute rhinitis and (*c*) ocular neuralgia, orbital, nasal or frontal. After giving details of the two cases mentioned, Charlin concludes that bacillary infection of nasal origin should be considered in all ocular disorders of obscure etiology and that this infection may exist without apparent signs, particularly in adults and especially in women approaching the menopause.

S. H. MCKEE. [ARCH. OPHTH.]

Miscellaneous

WHY VINCENT'S INFECTION? P. H. BELDING and L. J. BELDING, J. Am. Dent. A. **22**:2114 (Dec.) 1935.

This brief article summarizes the contention of the authors as expressed in previous publications. It is their opinion that trench mouth and pyorrhea, both acute and chronic, are caused "by the activity of Vincent's organisms (the fusospirochetal complex) and should be classified and treated accordingly." They recommend the following prescription, to be used with "surgical and mechanical cleansing": solution of potassium arsenite, 40 cc.; glycerin, 60 cc., and neoarsphenamine, 0.9 Gm.

NOYES, Chicago. [AM. J. DIS. CHILD.]

TREATMENT OF DISTURBANCES OF SPEECH AND OF THE VOICE. HUGO STERN, Wien. klin. Wchnschr. **48**:560 (May 3) 1935.

Stern discusses the treatment of the following disorders of speech: (1) dysarthria, in which a sound is spoken poorly, one sound is replaced by another or a sound is left out entirely; (2) stuttering; (3) paraphasia praeceps, in which speech is too rapid and whole syllables are often swallowed or confused; (4) late talking, a condition in which the intellect and hearing are normal but the child

does not want to imitate speech, as the motor speech center is not sufficiently excited by the hearing center; (5) nasalized speech; (6) inferior speech resulting from poor articulation and lack of tempo and melody; (7) hoarseness, which may be organic or functional; (8) paralysis of the recurrent laryngeal nerve, and (9) phonasthenia, or tiredness or weakness of the voice.

In regard to stuttering Stern states that it produces anxiety and the anxiety in turn makes the stuttering worse. No two cases of stuttering are ever exactly alike. Of utmost importance in treatment are the personality of the physician and the degree of rapport between physician and patient. The treatment must be started early, and there must be systematic exercises, though the patient must never be made too conscious of the fact that he is treated for stuttering.

ABT, Chicago. [AM. J. DIS. CHILD.]

HEREDITY OF SUBMUCOUS CLEFT PALATE. H. MAHLSTEDT, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **26**:347, 1935.

Mahlstedt designates rhinolalia aperta as the classic symptom of the submucous cleft palate. Depending on the size of the defect, the symptom is more or less severe. Examination of a patient with this symptom, particularly if the soft palate is raised, discloses in the hard palate a smaller or larger wedge-shaped defect which is covered with membrane. The uvula may be absent; there may be uvula bifida, or the uvula may be completely intact. It has been suggested that the submucous cleft palate represents incomplete spontaneous repair of the total cleft palate. Familial occurrence of the deformity indicates that it is a hereditary defect. The cases which are described by the author are of interest because the condition appeared in three successive generations. The diagram of the genealogical tree of this family indicates that the defect was transmitted to all members (two boys and one girl) of the first filial generation by a defective father in union with a normal woman. Two members of this filial generation (one brother and the sister) married healthy persons, and the defect appeared in three of five children in the one family and in two of three in the other. In all these persons with submucous cleft palate the uvula was missing, but the cleft in the palate differed in size. The author emphasizes that the defect was more severe in the family of the brother than in that of the sister. He points out, further, that submucous cleft palate, harelip and total cleft palate differ only in degree and that for this reason it is advisable to record the cases of submucous cleft palate in the genealogical trees of families in which such defects occur, even if the submucous defect is only slight. The author thinks that if this is done many of the genealogical trees will look quite different, and he hopes that more light will be thrown on the mode of hereditary transmission, so that it may eventually be decided whether the transmission is dominant or recessive.

EDITOR'S ABSTRACT.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Regular Monthly Meeting, Oct. 12, 1936

IRA FRANK, M.D., *President*

WALTER H. THEOBALD, M.D., *Secretary*

THE ONTOGENETIC APPROACH TO THE PATHOLOGY OF DEAFNESS. DR. LOUIS K. GUGGENHEIM, St. Louis.

Of the various approaches to the pathology of deafness, the embryologic has been the most neglected. The reason seems to be that few otologists understand the ontogenesis of the ear and embryologists are usually not interested in deafness.

An adhesive process or fibrosis of the tympanum was formerly considered to be entirely of inflammatory origin. If it was a sequel of chronic suppuration, it resulted from organization of granulation tissue or from direct differentiation of round cells into fibroblasts and connective tissue. In cases in which there was no suppuration a mild but long-continued round cell infiltration caused the fibrosis. The histologic picture of otosclerosis, accurately described by Politzer over fifty years ago, was accepted as the sole phase of that mysterious malady.

Then came a closer relationship between otolaryngology and general medicine and with it an army of doctors of philosophy telling otologists that everything they had accepted as pathology was but morphology, that in order to really understand disease processes they must recognize cytologic changes and explain the biochemical phenomena underlying them.

When in one case otitis media undergoes resolution with a return of normal hearing while in another it heals with marked impairment, one cannot be sure that deafness has resulted solely from toxemia of the labyrinth or from changes in the middle ear due to cellular infiltration and destruction of tissue. One cannot be sure that the unimpaired ear owes its escape to a less virulent invader or to a greater resistance by the host. When mastoiditis requiring surgical intervention develops from one diseased ear and not from another, there are, I believe, also some factors which have not been sufficiently emphasized. Otologists are beginning to realize that the structure of the ear itself may have a marked bearing on the outcome of disease.

Ears differ because of the varied morphologic expressions of genes and because of a multitude of environmental factors, such as the maternal vitamin, endocrine and general nutritional supplies to the developing fetus. An aural variable of great importance is the subepithelial mesenchyme of the tympanum and the mastoid antrum. Although described since the science of embryology came into existence, the biochemistry of the various differentiations of this pluripotent tissue has only recently begun to be disclosed.

Lateral projections of entoderm in the head end separate the mass of mesenchyme into the branchial arches. The evaginations of endoderm between the arches constitute the pharyngeal pouches. The first pair of these pouches become the eustachian tubes, the tympanums and the mastoid antrums. At first they are mere slits, but after the third month there is a rather rapid increase in the size of the tympanums owing to resorption of mesenchyme. At birth all tympanal and antral mesenchyme may have disappeared, or various amounts may persist throughout life. No norm has yet been determined. This tissue is soft and elastic and in no way interferes with ossicular movement or with any other function of the middle ear unless it is present in large amounts. Mesenchyme is present throughout the body, and tissue repair is dependent on its power of differentiation into connective tissue, new vessels, bone and other tissues. It is then one of the greatest of blessings, but in the middle ear it may prove a curse.

When the excess of mesenchyme has been resorbed from the tympanum and the antrum, the remainder differentiates into the thin tunica propria underlying the epithelium. When resorption is incomplete, leaving pads and bands of this tissue in the epitympanum, in the hypotympanum, about the ossicles and in the niches of the two windows, it becomes a menace in two ways. First, in the presence of infection with exudation, drainage and resolution are seriously interfered with. Second, the infection by calling forth the reparative potentiality of the mesenchyme may be followed by a rapid differentiation into fibroblasts and connective tissue. Permanent fibrosis with fixation of the stapes and other changes may result in lasting impairment of hearing. Fibrosis is then due not only to infiltrating new cells but to a transformation of existing tympanal tissue.

The microscopic differentiation between an adhesive process from unresorbed mesenchyme and one due to the formation of fibrous tissue from infiltrating inflammatory cells is easy in the early stages of infection but obviously impossible in advanced stages. In the former condition one notes beneath the cuboidal epithelium a broad layer of typical embryonic mesenchyme more or less infiltrated with polymorphonuclear leukocytes. Later some of the mesenchymal cells will have elongated into fibroblasts. In a more advanced stage collagen is beginning to form about the fibroblasts. In the latter condition, when the mesenchyme has been resorbed, there are in the early stages of infection a markedly swollen tunica propria, engorged vessels and many so-called Ruttin cysts. In addition, there is the usual leukocytic infiltration. Epithelium is destroyed in places. There is no evidence in the subepithelial spaces of mesenchymal syncytium but merely edematous connective tissue. In later stages fibroblasts from round cell differentiation are plentiful and the formation of collagen has started. Later still, when dense masses of fibrous tissue have formed, differentiation is no longer possible.

Only recently has the biochemical nature of the formation of fibrous tissue been disclosed. In connection with experimental scorbutus Wolbach and the Menkins showed that the secretion of intercellular substance—collagen—is impossible in the absence of vitamin C. In absolute scorbutus, collagen was made to form within twenty-four hours by giving the animal vitamin C. In absolute scorbutus the reparative response is active. Mesenchymal elements differentiate rapidly into fibroblasts, but the secretion of collagen fails. The new tissue has much the appearance of embryonic mesenchyme. Organization of a blood clot will begin, as usual, with migration of fibroblasts from adjacent tissue. In the clot the fibroblasts continue to multiply but without intercellular formation. Capillaries are unable to form for want of intercellular cement, but columns of endothelial cells appear. Marked osteoporosis occurs, and as matrix is lost the denuded osteocytes take on the appearance of fibroblasts.

The mensenchymal complex in otology then suggests the following questions: 1. What is the normal mesenchymal content of the tympanum and antrum at birth and later? 2. How can abormal amounts of mesenchyme in the middle ear and antrum be diagnosed? 3. How can one cause unresorbed mesenchyme of the tympanum and antrum to be resorbed before it is allowed to destroy hearing?

Normal and infected ears from birth to the age of 5 years are now being studied. The tunica propria is being studied under high magnification, and measurements are being recorded. Clinical histories are studied, and syndromes are being looked for, which may later be correlated with the histologic pictures. Certain clinical pictures even now suggest a possible relationship to unresorbed mesenchyme. These are: (*a*) suppurative processes of much longer duration than the usual, (*b*) recurrent otitis, (*c*) failure of the hearing to return to normal after infection of the middle ear, (*d*) a condition in which operation on the mastoid becomes necessary even though the general clinical picture is not one of virulent infection and (*e*) bilateral otitis when the process in one ear undergoes resolution in the usual time while the other continues to suppurate. Patients with such conditions often manifest, in addition, gastro-intestinal disorders, frequent nasal infection and malnutrition. Possibly in time certain syndromes will be recognized as accompaniments of unresorbed mesenchyme in the ears, and possibly the mesenchymal complex and the general picture will ultimately be found to be caused by a common metabolic factor. Such cases could then be recognized before

disease of the middle ear develops, appropriate treatment instituted and loss of hearing prevented.

The problem of promoting resorption of mesenchyme before infection and loss of hearing have occurred can best be solved by experimentation with animals. A recent observation of Lurie gives a valuable approach. Rats born without a pituitary gland have masses of mesenchyme in their middle ears and are deaf. Pituitary therapy causes the mesenchyme to resorb and the hearing to become normal.

(In a series of lantern slides two types of ear were followed from an early embryonic period to adult life. The first series demonstrated a normal process of mesenchymal resorption, and although infection of the middle ear occurred after birth, the final condition in adult life was normal hearing. The second series showed instances of failure of resorption of mesenchyme. Infection was followed by marked fibrosis and deafness.)

OTOSCLEROSIS

Little progress was made in the understanding of the pathogenesis of otosclerosis from the appearance of Politzer's reports of the histologic picture until 1931, when I reported the study of a 7 month fetus with an area of osseous dystrophy in the region of the round window and an abnormality at the site of predilection. A tentative diagnosis of the first stage of otosclerosis was made. After this Bast found, in several fetuses, areas of osseous dystrophy, which he thinks may be early phases of otosclerosis.

(After discussion of the merits of the various theories of etiology, the speaker's theory of regression was presented. Then followed a series of lantern slides showing the common, or second, phase of otosclerosis, the third, or quiescent, phase and the first phase, as seen in the 7 month fetus; finally a report was made of the two abnormalities which were found in a 6 week embryo with the classic pathologic picture of otosclerosis. These abnormalities are the absence of chondroblastic protoplasmic processes and the presence of red-staining spherules and fragments in the otic capsule. Cowdry's and Bartelmez' opinions of these abnormalities were reported. The abnormalities have been tentatively diagnosed as the anlage of otosclerosis.)

DISCUSSION

DR. B. J. ANSON: Evidently at least three important phases of otologic research are included in this interesting paper.

First, the management by chemical intervention of a fundamental process in histogenesis—more specifically, the limiting of tympanic fibrosis by alteration of the chemistry of living tissue. I am sure that those present have all followed the progress of these investigations with admiration and perhaps even with amazement. What delicacy in action would be required of the reagent utilized? It would be essential that the chemical action limit the production of excessive new collagen without liquefying the fibers of the stapedial ligament, the ossicular capsules and the submucosal and periosteal layers; it would also be essential that it in no way alter the related mesenchymal derivative, cartilage, since the stapes and the lining of the vestibular window are throughout life composed partially of cartilage. (Lantern slides were presented, showing the stapes at representative ages, from a 22.8 mm. embryo to a 70 year old adult, from Anson, Martin and Karabin: *Anat. Rec.* **64**:2-3, 1935.) The problem is apparently akin to that of limiting neoplastic tissue without destroying the host or seriously impairing the functions of vital organs.

Second, the inclusion of the fissula ante fenestram in the category of "vestigial structure." To determine definitely that it does or does not so belong would be to conduct a most engaging piece of morphologic research. I can report from a study of more than two hundred series of sections that it is definitely not represented by an epithelial structure in the embryos of mammals—cat, dog, rabbit, guinea-pig, rat, bat, pig, calf, sheep, deer and opossum (*Anat. Rec.* **59**:15-25, 1934). In general it must be remarked that in order to be reasonably certain that an observed structure in the shark or the ray is homologous with a certain one in the human body, it would be required that the particular structure be of widespread occurrence among the vertebrates belonging to the intermediate classes—

as was found to be true of villosities and kindred epithelial modifications of the lip (*J. Morph.* **47**:335-413, 1929).

As a hypothesis, adopted to guide an investigation, it may be indeed attractive, but as a theory, I believe that it is entirely presumptive.

Third, the explanation of otosclerosis as a retrogressive change, an expression of the body's attempt to reduce itself to a simpler and earlier ground plan—in this instance by obliterating, through the medium of a mesenchymal derivative, portions of a sensory organ essential to its well-being.

It is true, of course, that otosclerosis occurs so commonly in one place that the area is referrred to as the "site of predilection" (lantern slide), but new cartilage—never, so far as I know, destructive—likewise occurs there (lantern slide); moreover, the fissula may be incomplete even in the young. It may be crossed by bridges of bone; the fibrous tissue of the fissula may be continuous with the areolar tissue of neighboring marrow spaces. Partial obliteration by sclerotic bone is then only one of the aberrations it may exhibit; whether or not the latter must genetically succeed the formation of cartilage in an individual case I cannot yet say (Anson, B. J., and Martin, J., Jr.: Fissula Ante Fenestram: Its Form and Contents in Early Life, *Arch. Otolaryng.* **21**:303-328 [March] 1935). My associates and I do feel certain, however, that this area is "in a state of disequilibrium" histologically (Wilson, J. G.: *Acta oto-laryng.* **22**:382-389, 1935). But we cannot subscribe to the notion that this observed instability is due to the body's local tendency to rebuild itself into a less complex structure; that is contrary to the general trend of development; the adult human body does not lose ground phylogenetically.

Although we find it impossible to accept Dr. Guggenheim's theory, we are unable to offer one in its place. We expect an explanation of the causes of otosclerosis to follow on an extensive and unbiased study of many specimens.

Dr. Elmer W. Hagens: Dr. Guggenheim has presented an interesting and stimulating paper. I have always enjoyed talking these subjects over with him and hearing his views.

In regard to the mesenchyme tissue found in the ear I have noted the variations in this layer also. In a series of twelve children, varying from less than a year to 10 years of age, the thickness of the tunica propria or mesenchyme layer differed considerably. I have been surprised at its extent in a child, aged 3 years. We were able to see this condition to best advantage in the pneumatic spaces in the petrosa. In comparing children's bones with those of adults one often finds in the former a combination of the delicate mesenchyme plus the addition of the inflammatory reaction from infection. In adult ears the tunica propria is usually thickened immensely by only the results of the infection. If it is nature's way to combat an infection by a greatly thickened tunica propria, is it necessarily so harmful that the delicate mesenchyme persists? When the adult type of ear becomes infected, the thickened tunica propria is one of the outstanding findings. Therefore, in a short time the soft tissue thickening mechanically approximates that of a persisting mesenchyme.

I realize the difference between the delicate mesenchyme and a thickened tunica propria as the result of infection. However, could not the presence of the mesenchyme be even an asset in the presence of otitis media? It would seem to me that to understand these things one will have first to make sure of the presence of variations in retention of the mesenchyme by a study of a number of ears and secondly obtain adequate data as to the effects of such persistence of the mesenchyme. As. Dr. Guggenheim states, he and his associates are carrying on such research work at the present time. The experiments at Harvard are of interest, but from the bare facts mentioned one might question the interpretation of the findings. Perhaps the complete report of this work will give unqualified proof.

The regression theory of otosclerosis is certainly unique, but it is one of those subjects which at the present time can only be discussed and hardly proved. In clinical otosclerosis there is in some cases much more deafness than can be accounted for by the pathologic condition in the bony labyrinth. As Dr. Guggenheim has stated, several observers have reported changes in the nerve or the labyrinth. I should like to ask Dr. Guggenheim how he would correlate these cases

with his theory of regression. His statements regarding the site of the bony foci in relation to the more recently derived portions of the labyrinth is in keeping with what is found anatomically and clinically. I should rather state that otosclerosis is usually bilateral than that it is always so, as I have a specimen in which it is unilateral.

It is to be questioned whether when the capsule of the labyrinth is closed over by otosclerotic bone one can compare this situation with that in certain lower forms. It is still a human inner ear with a fully developed cochlea capable of hearing by bone conduction and not a fish ear with at most a lagena. The capsule of the labyrinth may have regressed, but the membranous labyrinth has not. Can one then make the comparison?

Dr. Guggenheim mentions the natural objection to this theory in stating that other examples of regression in man appear to be lacking. I should like to ask him more about the example of the sea squirt. Can one compare these changes in the larval stage with regression in a young human adult?

Dr. Guggenheim advised attacking the problem of otosclerosis through ontogenetic and phylogenetic investigation. I believe that study of the ears of fetuses for otosclerosis and whenever possible the study of the ears of other members of the family is of great value. The ability to produce the condition in animals should prove to be of the utmost importance and an attainment to be sought.

I hope that what I have said will be taken in the light of a thought-provoking discussion. Certainly Dr. Guggenheim's paper has caused me to do some thinking.

Dr. Francis L. Lederer: I do not wish to place myself on record as discussing the theory. It has been amply discussed. I do not believe that any one can dispute the evidence presented by the slides Dr. Guggenheim has shown, even though he may not be in full accord with his interpretation or his theories. The members of the society have been well repaid for their attendance this evening. Ordinarily a large attendance is not to be expected unless the speaker is to produce evidence of successful treatment. However, the members appreciate this type of work and are aware of the immense effort given to it. I am grateful to Dr. Guggenheim for his presentation. I cannot add anything to what the previous discussers have said. As a matter of fact, the reason they were placed on the program was that they might adequately represent the society.

Dr. George E. Shambaugh Jr.: I should like to ask Dr. Guggenheim to explain the cases of deafness due to persistent mesenchyme in the middle ear and should like to know how he differentiates those from other cases of deafness of the middle ear.

Dr. Alfred Lewy: I think that this is one of the most interesting and broadest investitgations of this subject that has ever been presented before this or any other society. This, together with the philosophical summing up, is a gem. I shall not attempt to discuss the subject. There is, of course, a great gap in it, as shown by Dr. Anson, but it is a gap that may be bridged by further investigation. Perhaps the archeologists may find some phylogenetic approach or bridge to substantiate the philosophic part of his discusison. He mentioned in connection with fibrosis the relation to vitamin C. Is there any evidence that these children who present frequent otitis media and other evidences that are said to be traceable to the nonabsorption of the mesenchyme are of the so-called scorbutic type that physicians now talk about in trying to diagnose what is called subclinical scurvy? If so, would it not be feasible to attack the problem on this basis?

Dr. Robert Sonnenschein: Only two things occur to me at the present moment. The first is my ignorance of the subject, and the other is my unbounded admiration for Dr. Guggenheim, whom I have known for many years. I have heard him present the subject before but never so completely as tonight. It does seem that in attacking a problem from the standpoint he has taken he may finally be able to give an idea of the etiology of otosclerosis, and if that occurs there may be a possibility of doing something clinically, and that all otologists hope for.

Dr. Louis K. Guggenheim: I am grateful for the discussion, from which I have profited much. To Professor Anson I would say that I am aware that my theory of otosclerosis has proved nothing; it has, however, accomplished something of real value. A few years ago when research on the problem of otosclerosis was almost at a standstill, the embryologic and phylogenetic investigation insti-

tuted at the Oscar Johnson Institute gave new impetus to research on deafness. A vast new territory of investigation was opened up. Since then the work on the fissula ante fenestram carried on by Dr. Anson at the Northwestern University and Dr. Bast at the University of Wisconsin has aroused a tremendous new interest in the anatomy of the temporal bone and the problem of otosclerosis. After my discovery of the osseous dystrophy in the 7 month fetus, Bast a few months ago reported several examples in his specimens.

As for the theory of the origin of the fissula, again I am fully aware that the existence of the accessory endolymph structure of the shark and the ray and the canalis fovea fenestrae ovalis of Rana esculenta do not prove the vestigial nature of the fissula but the finding of these structures in the lower animals is certainly a lead for further study of the structure in man, which apparently has no function. In this connection it is highly important that the finding of the fissula in the hyena and antelope by Hyrtl should be substantiated by further study of these two animals. If any of those present happen to have a hyena or an antelope, I should be most grateful for its ears, which thus far I have been unable to obtain.

As for Dr. Hagens' question as to the relationship of the osseous changes in otosclerosis to involvement of the nerve. I can say only that I have not been able to observe the changes of the nerve described by Gray and do not know the explanation of the involvement of the nerve in otosclerosis unless it is due to pressure from a focus at the internal meatus. I do not even know what the nature of the involvement is, for when I do find in conjunction with otosclerosis atrophy of the spiral ganglion, stria or other nerve tissue I cannot be sure that the changes are not independent ones which occur without otosclerosis.

Dr. Hagens doubts that the persistence of mesenchyme and the transformations of this tissue can be such a bad thing. Although my research has not progressed to the point at which I can speak with certainty, I believe that in a large percentage of the cases deafness is due to the differentiation of unresorbed mesenchyme into fibrous tissue.

Dr. Hagens asks: Why, if the theory of regression is true, does the capsule show the regression while the membranous labyrinth does not? I can only answer that I am not sure that the involvement of the nerve in otosclerosis is not a regression also. Further, if it is proved that the capsular changes alone constitute otosclerosis it may be because the capsule is derived from mesenchyme while the membranous labyrinth and nerve structures are of ectodermal origin and perhaps a reason will some day be found for the regressive changes in the one and not the other. In the ascidian the brain, eye and ear regress while the remainder of the animal persists as a sessile organism.

I am aware that a comparison of the regressive changes in the ascidian to a human malady in my address is far-fetched; however, when one is making a phylogenetic investigation to throw light on a human problem one cannot exclude any phylum no matter how low its position in the scale of evolution is.

Dr. Shambaugh asks for the typical clinical picture of the patient with unresorbed mesenchyme in the middle ear. I stated that such a picture is not known but that certain syndromes commonly observed suggest even now a relationship to unresorbed mesenchyme. I hope in time, through clinical study correlated with histologic observations and experimentation with animals, finally to become able to recognize the condition before hearing is lost.

I am much interested in what Dr. Lewy has said about the early recognition of scurvy in children, but I am afraid that treatment for scurvy—addition of more vitamin C to the diet—would be a "scurvy" trick on the ears with unresorbed mesenchyme, as innocuous mesenchyme can only be aided in its transformation into fibrous tissue by the addition of more vitamin C. If one could give the ears scurvy and make the rest of the body normal, it might work for a better result.

What I have tried to present tonight is a new avenue of approach to an old and hitherto hopeless condition, fibrosis of the tympanic cavity. As for otosclerosis, my theories have proved nothing, but again they have opened up a new field of research which has already borne fruit, as witness the important embryologic investigations of Anson and Bast.

I appreciate the patience of the audience and the valuable discussion which followed my presentation.

Book Reviews

Diseases of the Air and Food Passages of Foreign-Body Origin. By Chevalier Jackson, M.D., and Chevalier L. Jackson, M.D., Philadelphia. Price, $12.50. Pp. 994, with 2,000 illustrations, including 3 plates in color. Philadelphia: W. B. Saunders Company, 1936.

This comprehensive treatise on the subject of foreign bodies in the respiratory and food passages consists of 994 pages. In separate chapters the authors deal with the etiology of foreign bodies in the air and food passages, the prophylaxis, pathology, symptomatology and diagnosis of foreign bodies in the air passages, treatment of patients with foreign bodies, mechanical problems of removal, extraction of foreign bodies from the esophagus, diagnosis and the treatment for foreign bodies in the gastro-intestinal tract. Altogether, 333 pages of the treatise are devoted to these subjects. An appendix including elaborate tables comprises the remainder of the volume.

The treatise will be of interest to the general practitioner as well as to the specialist in this field.

News and Comment

THE AMERICAN BOARD OF OTOLARYNGOLOGY

An examination was held in New York on Sept. 25 and 26, 1936, prior to the meeting of the American Academy of Ophthalmology and Otolaryngology. Of the 120 candidates examined, 103 were certified.

An examination will be held in Philadelphia on June 7 and 8, 1937, during the Annual Session of the American Medical Association in Atlantic City, N. J.

Prospective applicants for the certificate may secure application blanks from the secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha.

Directory of Otolaryngologic Societies *

FOREIGN

COLLEGIUM OTO-RHINO-LARYNGOLOGICUM AMICITIÆ SACRUM

President: Prof. Gunnar Holmgren, Karolinska Mediko-Kirurgiska Institutet, Stockholm, Sweden.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg, 143 Groningen, The Netherlands.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

Chairman: Dr. Robert F. Ridpath, 1737 Chestnut St., Philadelphia.
Secretary: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Place: Atlantic City, N. J. Time: June 7-11, 1937.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Frank E. Burch, 408 Peter St., St. Paul.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Detroit.

AMERICAN BRONCHOSCOPIC SOCIETY

President: Dr. Joseph C. Beck, 185 N. Wabash Ave., Chicago.
Secretary: Dr. Lyman Richards, 319 Longwood Ave., Boston.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. W. B. Chamberlain, 10515 Carnegie Ave., Cleveland.
Secretary: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Hotel Traymore, Atlantic City, N. J. Time: June 3, 4 and 5, 1937.

SECTIONS:

Eastern.—Chairman: Dr. D. C. Jarvis, Quarry Bank Bldg., Barre, Vt.
Place: Boston. Time: Jan. 7, 1937.
Southern.—Chairman: Dr. Charles D. Blassingame, P. & S. Bldg., Memphis, Tenn.
Place: Memphis, Tenn. Time: Jan. 13, 1937.
Middle.—Chairman: Dr. Alfred Lewy, 25 E. Washington St., Chicago.
Place: Chicago. Time: Jan. 11, 1937.
Mid-Western: Dr. Frederick A. Figi, 436 Tenth Ave., Rochester, Minn.
Place: Chicago. Time: Jan. 11, 1937.
Western.—Chairman: Dr. David R. Higbee, 3245 Fourth Ave., San Diego, Calif.
Place: San Diego, Calif. Time: Jan. 30 and 31, 1937.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. Edmund Prince Fowler, 140 E. 54th St., New York.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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